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The Philadelphia Neurological Society and
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VOLUME 30, 1903

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Vol. 30 January, 1903 No. 1

# The Journal of Nervous and Mental Disease

The Official Organ of

The American Neurological Association
The New York Neurological Society
The Philadelphia Neurological Society and
The Chicago Neurological Society

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Printed and Published by the Alliance Press Company, 38 W. 18th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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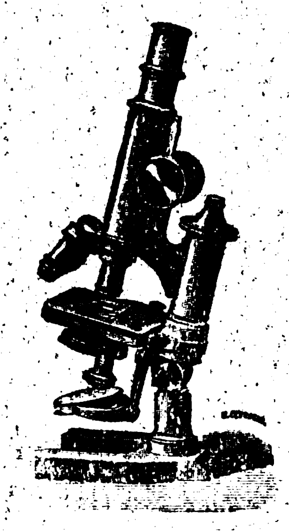

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THE
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## Original Articles.

# ON AMAUROTIC FAMILY IDIOCY.
## A DISEASE CHIEFLY OF THE GRAY MATTER OF THE CENTRAL NERVOUS SYSTEM.[1]

By B. Sachs, M.D.,
PROFESSOR OF MENTAL AND NERVOUS DISEASES IN THE NEW YORK POLYCLINIC, *etc.*

The clinical symptoms of amaurotic family idiocy have been described frequently and thoroughly enough. It is gratifying to note that the clinical picture as developed by the present writer a number of years ago has been generally accepted, and has been fully endorsed in the excellent articles by Hirsch, Peterson, Mohr, Patrick, Falkenheim, and a number of others. Two symptoms, in addition to those originally given, have been mentioned by Falkenheim:—"Explosive laughter" and "disturbance of deglutition." The former I have not observed, though I do not doubt its occurrence in some instances, and the latter may be taken to be either an accompaniment of the great mental and physical deterioration, or it may, at times, have the value of a "bulbar" symptom; and "bulbar" symptoms are explained easily enough, in view of our present knowledge of the morbid anatomy of the disease.

While the disease is relatively rare, the records have become so numerous that Falkenheim has been able to analyze 64 cases.* The number is increasing rapidly and mere statistical studies will soon not be needed. I would urge, however, that the clinical type as now accepted, be not disturbed because of any unusual symptoms, such as were recorded, for instance, in the cases of Claiborne, Kuh and De Bruyn. The last named author has, of his own accord, and justly so, withdrawn his case from this especial type. In the case of Claiborne the condition was complicated by a tubercle of the corpora quadrigemina, and although the characteristic change in the fundus was made out, the patient presented so many unusual features that this record should be utilized, if at all, with great caution. In Kuh's case the clinical picture was obscured and altered by the presence of an hydrocephalus.

---

[1] Read at the annual meeting of the American Neurological Association, June 5, 6, and 7, 1902.

*I have another case under observation at the present time.

The chief interest in the study of amaurotic family idiocy has been shifted from the clinical to the anatomical side. There is, in the main, a fortunate agreement as to the findings. There can be a difference of opinion only with respect to the interpretation to be put upon these findings. Nine years have elapsed since I have had the opportunity of studying carefully the morbid changes in a case that had been under my own observation. Since that time so many new staining methods have been introduced that I was eager to undertake this special study in pathology again, and to determine to what extent conclusions of former years would have to be changed or reinforced. For the opportunity of studying the present case I am greatly indebted to Dr. Sarah Welt, who devoted an unusual amount of time and patience to the care of this child before finally referring it to Mt. Sinai Hospital; and I am also indebted to Dr. Koplik, who, knowing my interest in the subject, was good enough to put this child entirely under my charge. As in all the other cases, the history of this one may be stated briefly:

This child, C. S., born of Hebrew parents, was two years and seven months of age at the time I first saw it at the hospital, in September, 1901. The patient's mother had had three children. The first-born is now six years of age, a fine, healthy-looking child. The second child died at the age of sixteen months. Two months before its death the mother knew that it was blind. It could never sit up straight; never uttered a sound; had no spasms, but was feverish off and on. It was thought to have tubercular meningitis, but in all probability was afflicted with this family disease. Our own patient, the third child, was born about two and one-half years after the second. During pregnancy the mother was nervous, but otherwise well; labor was normal and short. At birth and for some time afterward the child appeared to be normal; it vomited a great deal, however, and as time passed on it was noticed that it could not sit up straight, nor did it seem to be as bright as other children. The mother thought that the child recognized her when it was somewhere between six and ten months of age, but after this period the vision gradually grew fainter and fainter. At the age of one year the child was practically blind, and since then the general weakness increased. At about this time the typical changes in the region of the macula lutea were made out. At the age of nineteen months it had a vomiting spell with convulsions. In November, 1900, it was afflicted with pneumonia, from which it recovered, but the mother states that thereafter it never moved its extremities except in convulsions. The extreme contractures in the upper and lower extremities which the child presented were developed about February, 1901. The statement was made that these came on quite suddenly after a convulsion, but of this fact we cannot be entirely certain. The child had difficulty in swallowing and was startled by the slightest noise. With the exception of increasing emaciation, the condition remained practically unchanged up to the time of death, on October 16, 1901.

The history as given above is largely based upon the careful observation of Dr. Sarah Welt, and the condition was also carefully studied by me during the four weeks preceding death. In the last two weeks of life the temperatures were often subnormal. To complete the history of the case, it may be added that the mother nursed this child to the age of eleven months. She had nursed the second child to the age of eight months, while the first healthy child was raised on sterilized milk.[2] While carrying the amaurotic child, the mother felt very little life, and in passing it may be noted that she was distinctly of cachectic build. From the above account of the child's condition it will readily be seen that the diagnosis of amaurotic family idiocy could be made with absolute certainty.

The autopsy was performed ten hours after death, on October 16, 1901, by Dr. Libman, Assistant Pathologist of Mt. Sinai Hospital. To him and to his chief, Dr. Mandelbaum, I am indebted for a full report. We could not get permission to remove the eyes, but with this one exception the study is complete.

Upon removal from the skull the brain weighed 784 grammes; the dura mater was rather firmly adherent to the bones.

---

[2] I make mention of these facts in order to do justice to Hirsch's theory. The facts would seem to support his theory, but for reasons to be given later on I cannot accept the same.

The cortex was so firm that the knife grated as it passed through it—a sound which I have not heard under similar conditions since the autopsy on the first subject of this disease. It was noted that the convolutions were small and that there were fissural peculiarities,—indications of a low type of brain, such as I recorded in the very first report made upon this disease, but such changes have not been referred to in the reports of others. In view of the possible developmental character of the disease these structural peculiarities are of some importance. The fluid in the pia was markedly increased in amount. The pituitary body was not enlarged.

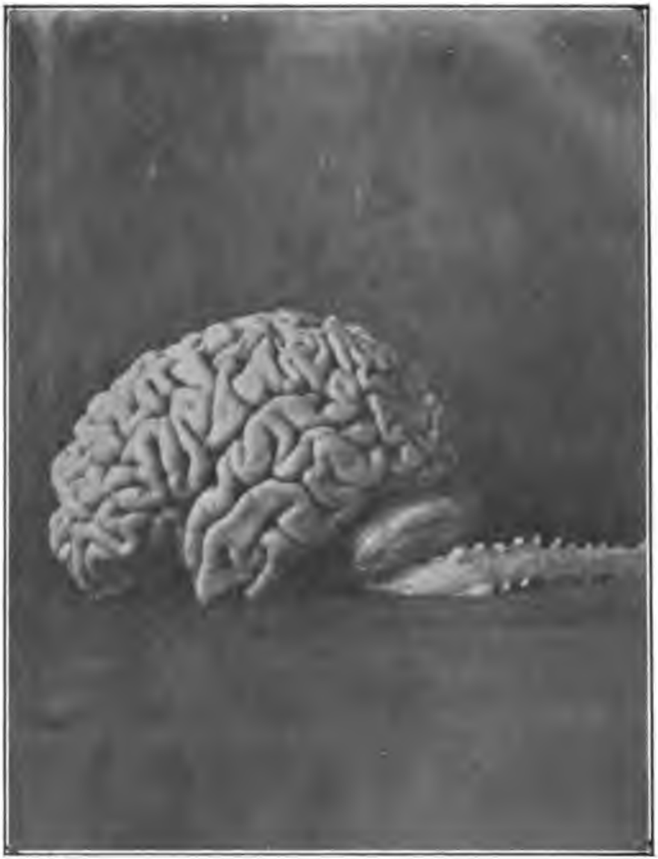

Fig. 1

Of other changes noted at the time I will only refer to an old pleurisy, and to a very advanced parenchymatous myocarditis.

The brain and spinal cord were at once put in formalin and prepared for staining by the various methods now in use. Serial sections have been laid through almost every part of the entire central nervous system. A careful study of these sections shows some deficiency in the development of the cerebral white fibers and also a degeneration of the pyramidal tracts in the lateral as well as in the anterior columns of the cord. This same degeneration can be traced through the course of the pyramidal tracts in the internal capsule, crusta, pons and medulla, but much more striking than this change in the white fibers is the change in the gray matter of the central nervous system. The changes are found to be the same in the cortex of the brain, in the cranial nerve nuclei, in the anterior and posterior gray matter from the cervical to the lowest lumbar, and sacral segments of the cord. Even the sacral

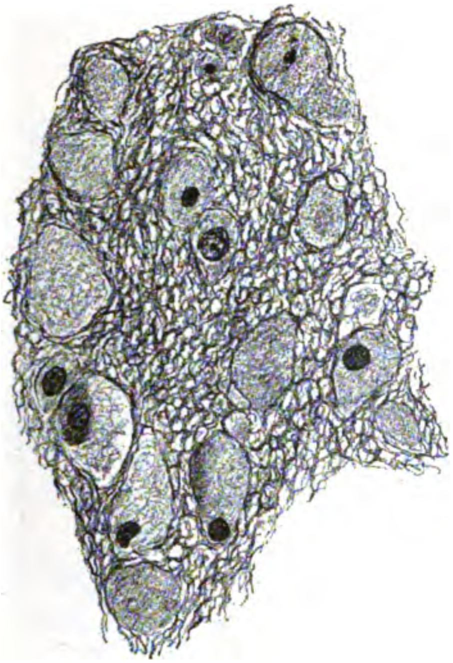

Fig. 2—Section through cortex, showing changed pyramidal cells. Hematoxylin-eosin stain. Oc. 4; obj. 1-12 (Leitz).

ganglia show similar changes. The changes are practically those to which attention was explicitly directed by Dr. Hirsch, and which were found to be similar to those reported by me in the cortex in 1887, and were also found on a reëxamination to be contained in some of the spinal sections of the specimens examined in 1892 from my second case.

Of course these changes are brought out much more beautifully by the Nissl and other methods now in use. There is unquestionably some increase of the neuroglia cells throughout the central nervous system, but the chief and most remarkable changes are found in the larger ganglion cells throughout the entire extent of the central nervous axis. In the cortex as well as in the anterior gray matter of the cord there is not a normal ganglion cell to be seen. As will be noted in the specimens and illustrations here-

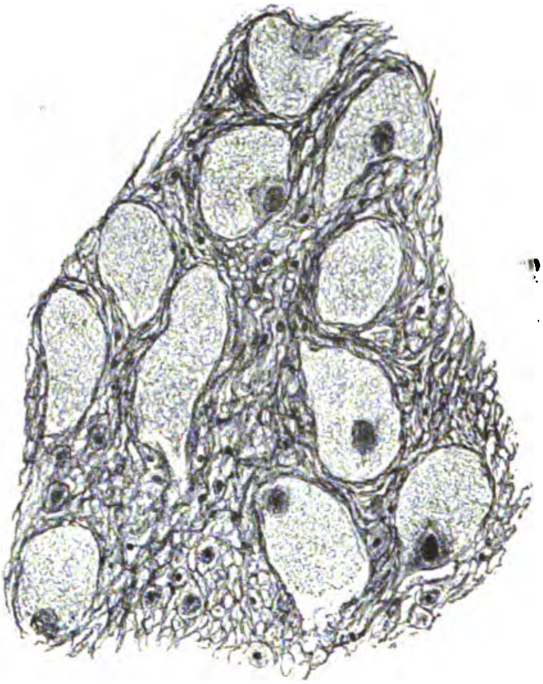

Fig. 3—Section through XII nucleus in medulla oblongata showing advanced changes in ganglion cells. Hematoxylin-eosin stain.

with presented the cell body is completely altered, the entire cell protoplasm has become disintegrated, leaving a more or less homogeneous mass, the nucleus has been shifted commonly to some part of the periphery of the cell, and instead of being distinctly differentiated from the rest of the cell-body it generally shades off by degrees into this aforesaid homogeneous mass. The nucleus is often wanting entirely, and, in some instances, the entire nucleus appears to have dropped out. The disintegration of the cell-body is so complete that in many specimens the contour alone enables one to infer that these round bodies are the remnants of ganglion cells. These metamorphosed ganglion cells are frequently surrounded by distinct pericellular spaces and are relatively and absolutely so much enlarged that they give a very striking appearance to the cross section.

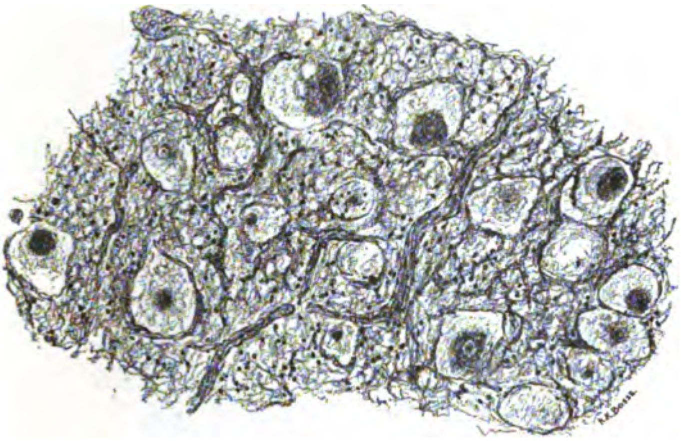

Fig. 4—Section through cervical enlargement showing cell changes in anterior gray matter. (Van Gieson stain.)

There is no doubt an entire agreement between the findings in this last case of mine and those so well reported by Dr. Hirsch before this Association a number of years ago. Curiously enough, other recent writers have not been struck by these changes in the gray matter of the brain and spinal cord. In a recent and preliminary report by Prof. Schaffer of Buda-Pest there is the account of a typical case and in the report of the post-mortem findings special stress is laid upon changes of the white fibers and nothing is said about the gray matter, except that it is entirely normal, and yet, from the cut (Fig. 2) which accompanies his article, although it is reproduced at a low power, I suspect that these same large cell-bodies are present, such as Hirsch and myself have found and described.

The chief inference to be drawn from a study of sections laid through every part of the central nervous system in this and in other cases of amaurotic family idiocy is that the morbid process in this disease affects primarily, or at least to a great extent, the entire gray matter of the brain and of the spinal cord. The degeneration of the white fibers of the anterior and lateral pyramidal tracts is in all probability secondary, but not nearly so great or so marked as one would expect with such advanced disease of the ganglionic tissue. It is already probable that the relative involvement of the gray and white matter may vary in different subjects of this disease.

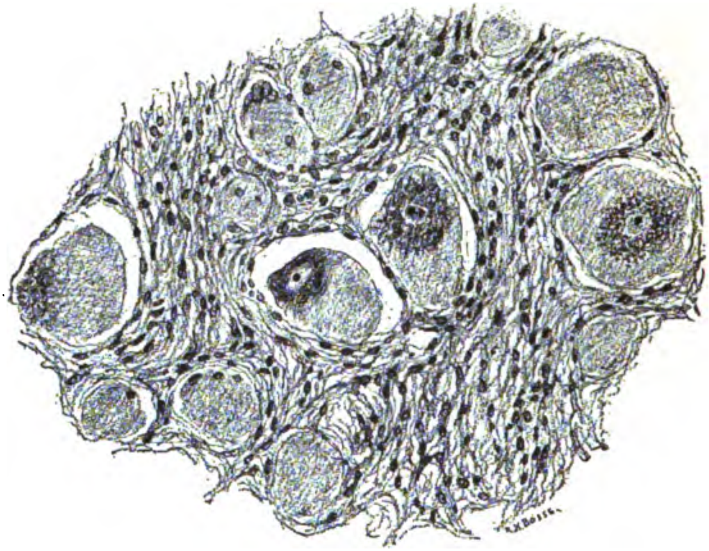

Fig. 5—Section through a lumbar ganglion. Nissl stain showing marked cell changes.

The neurone theory has received so many severe blows of late that the somewhat anomalous condition of affairs as observed in this case need not be especially dilated upon. The theory may have to go to the wall in part or as a whole, but the facts will stand and will call for further elucidation.

In 1887 I spoke of these changes as representing an agenetic condition pure and simple affecting the highest cortical nerve elements. At that time I was not aware of the widespread character of the morbid process. Later researches, and particularly those of Kingdon and Russell and of Hirsch, have laid stress upon the degenerative character of the disease. Much has been made of the fact that the present author has recorded the process as essentially an arrest of development, while the other writers just mentioned have insisted upon the purely degenerative and acquired character of the disease. Some writers have accepted these expressions of opinion as though they indicated a widely differing conception of amaurotic family idiocy, but the difference between the views of the authors just mentioned and of myself is not so great as it would appear at first sight.

A degenerative process does not necessarily represent an acquired or an acute affection. It is my contention now, as in former years, that degeneration will set in whenever normal growth is arrested, but life continues. A child to be afflicted with amaurotic family idiocy is born with a limited and restricted capacity for normal development. Its gray cells may do as well as those of any other child up to the age of two or three or six months, but beyond that its powers for further development will not go. To this extent, in this respect, the disease is after all a congenital affair; when normal development ceases, degeneration sets in.

Gowers has recently written a very helpful article in which he applies a similar course of reasoning to the various scleroses, and has suggested the term "abiotrophy," which I gladly accept, to indicate this arrest of normal development followed by degeneration. The various parts of the nervous system, according to Gowers, have their own vitality. "Some of them may slowly die, while the life of all the rest goes on without impairment. * * * When the failure is early it is due only to a defect which seems to be inherent, the tendency thereto inborn. We do not indeed apply the word 'death' to this slow decay of the elements; we speak of it as 'degeneration'; but the process is in many cases—perhaps in most—an essential failure of vitality." The term "abiotrophy" designates a lack of vital nutrition, while the term "abiotic" would designate that which depends on defective vitality. Were we to adopt Gowers' phraseology, we might with good reason claim that amaurotic family idiocy is due chiefly to a defective vitality of the gray matter of the central nervous system.

To my mind there is an almost insuperable objection to the doctrine that so distinct a family affection is an acquired disease; yet granting the possibility of this it could only be conceivable upon the further theory of toxic or other influences acting upon the tissues of deficient embryonal potentiality. Hirsch's contention that the toxic influence may have been exerted through the mother's milk cannot be maintained for the simple reason that several of my patients have not been nursed by the mother. Assuming that amaurotic family idiocy may be an acquired disease, the toxic nature of the degenerative process has been argued from the resemblance of the cell pictures to those found in cases of poisonings of various kinds. This point deserves careful consideration, but cannot be decided unless we are certain that the cell changes as we see them represent definite changes due to definite causes.

With the refined staining methods of the day, we have learned much regarding the protoplasmic changes of the cell-body. But I ask, does acute poisoning produce cell changes distinctly different from those of chronic poisoning? Again, can we make out from inspection of the cells whether death was due to metallic poisoning, to acute infectious disease, to a condition of marasmus or what not? For the present it would be hazardous to present such a claim.

It would seem more profitable to attempt to discover other conditions in which cell changes occurred somewhat similar to those made out by Hirsch and myself.

Dr. Spiller reported the case of a boy about nine years of age, the inmate of the Pennsylvania Training School for Feeble-minded Children, who was in all probability afflicted with congenital spastic rigidity and imbecility. This child was seized with symptoms resembling those of cerebro-spinal meningitis from which he died five days after the onset. The microscopical examination of the nervous tissue, Spiller writes, showed little evidence of inflammation.

"I found cell changes in all the cell-bodies of the posterior and anterior horns of the spinal cord; in the nuclei of the cranial nerves; in the sensory as well as the motor nucleus of the fifth nerve; in the cells of Purkinje and in the parietal lobule where especially the cells of Betz were altered. These cell changes were very like those found in amaurotic family idiocy. The gross lesions of meningitis were not found." Spiller seems to interpret these cell changes to be the result of the acute process. I am tempted to question him whether they might not more properly be related to the "chronic spastic rigidity and imbecility." Are we certain enough of these cell changes to claim that they must necessarily be the product of some acute intoxication?

The findings of Rolly in a case of Little's disease (congenital spastic diplegia) are of special interest in this connection. The child was the second of twins, born in the mother's fourteenth confinement. The child was born asphyxiated, and presented all the symptoms of marked spastic rigidity (diplegia), with frequent convulsions. These symptoms continued without much change until death, at the age of about two and one-half months. Rolly lays stress on the specific history of the father, and wishes chiefly to prove the bearing of syphilis upon the etiology of Little's disease. For my present purposes I am interested in his cell findings, Nissl himself having examined Rolly's specimens taken from the spinal cord and from the brain. In the lumbar portion of the cord the author states: "Side by side with cells which present a tolerably normal appearance are others which exhibit a granular disintegration of the Nissl bodies surrounding the nucleus, while the peripheral portions of the cell body remain practically normal." Similar changes were found in the dorsal portion of the cord. In the cervical portion "there are fewer normal cells than in the other divisions of the cord. As a rule, the nucleus cannot be distinctly differentiated from the cell protoplasm. More marked still are the degenerative changes in the brain. There is an enormous and diffuse proliferation of the neuroglia. Some ganglion cells have lost their contours and are more or less shapeless masses, which shade off into the surrounding tissue. Some ganglion cells are so thoroughly disintegrated that one sees practically only lacunæ, which, by their shape, remind one of the ganglion cell form. Small vacuoles were also seen to be present in some of the ganglion cells."

All of these changes bear the closest resemblance to the findings in amaurotic family idiocy. Had this child lived to the age of the amaurotic children previously described, the change would no doubt have been still more striking.

I would also call attention to similar findings in the cases of "generalized rigidity" as described by Collier,[a] and in a somewhat similar case reported by Mya and Levi. Whether these latter cases are described as spastic diplegias or as cases of "spastic pseudobulbar paralysis," according to Peritz, the point that is of special interest to us is that very similar anatomical changes have been found in the brains of children who had been afflicted with varying forms of cerebral disease.

Peritz is inclined to the opinion that I have made a special effort to establish amaurotic family idiocy as an entirely independent disease. In this he is mistaken, for I have contended for nothing more than that this family disease represents a distinct and easily recognizable clinical type. This is evident from the fact that in this city not only neurologists, but pediatrists and general practitioners have learned to diagnosticate amaurotic family idiocy with surprising ease on clinical grounds. The disease can be differentiated easily from the other forms of idiocy and from the various types of infantile cerebral palsies. In his earliest publications on this subject the present writer endeavored to show the relationship between amaurotic family idiocy and other congenital cerebral conditions, but he considered it necessary to proceed cautiously before arriving at any definite conclusion. At the present time, however, in view of the researches of Hirsch, Rolly, Spiller, Collier, Peritz, Sachs and others, it may definitely be stated that there is a close anatomical relationship between amaurotic family idiocy and other cerebral diseases of childhood which are dependent on an arrest of, or at least a disturbance in, the normal development of the central nervous system.

I will not as yet claim that the morbid process in all of these children is identical, but it seems to me that the only just inference to be drawn is this, that in amaurotic family idiocy the entire central gray matter is the seat of an intense degeneration, and that somewhat similar changes are found in conditions more or less closely allied to this special type of family disease. But even if closely allied to other affections, the clinical picture of the type is so distinct that we can well afford to consider it a special clinical form of family disease. Why children of one race should be afflicted so much more often than those of others, when the allied conditions show no such preference, remains as great a puzzle as ever.

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[a] Collier calls attention to the resemblance between his anatomical findings and those of amaurotic family idiocy.

We have not yet reached the end of this discussion, and I trust that with every new case carefully reported and carefully studied further light will be thrown upon a disease that appears to me to be of increasingly greater interest. Above all things, I would have future writers on this subject pay the closest attention to the changes in the gray matter of the brain and of the spinal cord.

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# THE POSTERO-LATERAL SCLEROSES[1].

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We report eight cases of sclerosis of the posterior and lateral columns of the spinal cord, and propose to place them as far as is possible in a classification of combined diseases of the cord. The first four are cases of degeneration of the cord associated with anemia. The history of the first, one in which the anemia was secondary to chronic parenchymatous nephritis, is as follows:

Mrs. G., 46 years old, a widow, was admitted to the Home for Incurables, 7, 26, '01. Her father and one brother had died of carcinoma, her mother and two brothers of phthisis. She had never been pregnant. She had had typhoid fever with pneumonia eight years before. Except for this illness her health had been good. Four years before her admission to the Home her legs began to be weak, and she could not walk as well in the dark as in the light. After several months of rest she improved sufficiently to return to her work. She remained in fairly good health, but always with some weakness in the legs until last year, since which time she has been growing steadily worse. She now complains of great weakness in the legs, and of a sensation of "misery" in them, but not of pain. She says that at times the legs jerk and twitch.

When she is helped to walk the gait is very ataxic. Static ataxia is also very marked. There is no muscular wasting. All the extremities are weak. There are irregularly-shaped scattered areas of anesthesia on the thighs and legs. Sensation is delayed and often doubled on the feet. The knee-jerks are absent. Sticking either side causes extension of the toes. The reflexes of the arms are normal. The urine contains a small quantity of albumin and granular casts. The blood examination showed H. 30 per cent.; R.B.C., 1,280,000. Her condition gradually grew worse. In September of 1901, the kidney trouble became acute, the anemia pernicious in type, and she died of uremia. Just before death the red blood cells numbered 980,000, and the hemoglobin estimation was 28 per cent.

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[1] Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

The necropsy revealed chronic parenchymatous nephritis, chronic gastric catarrh, and the usual changes in the bone marrow of the pernicious type of anemia. The brain was macroscopically normal. Slight chromatolysis of Betz's cells was present. The spinal cord showed diffuse degeneration, especially in the posterior and lateral columns in which parts it was associated with much sclerosis.

The cervical cord showed in the posterior columns dense sclerosis resembling secondary ascending degeneration. In the postero-external columns the degeneration was less marked and tended to reticular formation. The lateral columns showed dense sclerosis of the crossed pyramidal, less dense of the cerebellar tracts, and a diffuse degeneration of the antero-lateral and direct pyramidal tracts. In the anterior part of the cord there was a slight marginal sclerosis such as is frequently seen in senile cords. The glial cells around the central canal formed a mass at least twice as large as usual. The anterior horn cells examined by the Nissl method appeared normal, but there was a very large amount of yellow pigment, in some cases filling the entire cell. Examination by the Marchi method showed degeneration, with black clumps and compound granule cells in the posterior and lateral columns. There was well defined degeneration in both the anterior and posterior roots. The pigment in the anterior horn cells stained black where treated by this method.

In the dorsal region of the cord the crossed pyramidal and direct cerebellar tracts and the posterior columns, except the root zones, were densely sclerotic. The remainder of the white matter was diffusely degenerated with hollow spaces here and there. The anterior horn cells resembled those in the cervical region. The central canal was packed with and surrounded by a glial formation which also involved both commissures.

In the lumbar cord there was slight sclerosis with hollow spaces in the posterior median columns, and a few hollow spaces in the posterior external columns. There was slight sclerosis of the crossed pyramidal tracts. The remainder of the white matter showed very little change. The proliferation of glial cells around the central canal amounted to a distinct tumor formation, triangular in shape and extending backwards into the posterior columns and laterally into the gray matter of the median portion of the anterior horns.

To sum up: there was diffuse degeneration of the white matter of the cervical and dorsal areas with dense sclerosis of the postero-median columns and crossed pyramidal tracts throughout the cord; degeneration of the anterior and posterior roots, shown by the Marchi method; marked central gliosis most marked in the lumbar and least distinct in the cervical region; slight marginal sclerosis; no meningeal lesion.

We are enabled to report the second case through the courtesy of Dr. D. J. Milton Miller. The patient, J. L., a woman, sixty years old, complained for two years of numbness and tingling in the hands and forearms especially after they had been in hot or cold water. There was nothing of importance in the family or previous personal history. The clinical course of the disease while the patient was in the hospital was that of pernicious anemia. On admission the blood examination showed 27 per cent. of hemoglobin and 1,000,000 red corpuscles. A short time before death the hemoglobin had increased to 47 per cent. and the red corpuscles to 1,800,000. The reader is referred to Dr. Miller's paper for details of the clinical history. The only nervous symptoms were shooting pains in the legs, a well defined girdle feeling, numbness in the arms and legs, loss of power and impaired sensibility in the right arm and hand, delayed sensation in the legs and the absence of all the reflexes in the legs.

The patient died on January 3, 1902. Examination of the brain revealed only a slight diffuse chromatolysis of the large ganglion cells of the cortex. In the spinal cord sclerosis was well marked in the crossed pryamidal and direct cerebellar tracts, less marked in the antero-lateral and Gowers' tracts. Slight areas of degeneration existed in the direct pyramidal tract and anterior ground bundle. The median portion of each posterior column showed dense sclerosis. The remainder of the posterior columns showed a diffuse sclerosis with a large number of myelinated fibers intact. These fibers were arranged crosswise, some running transversely, others in whorls.

In the dorsal cord the degeneration and sclerosis affected the entire posterior columns except the root zones. A diffuse sclerosis affected the lateral tracts and a number of hollow areas were scattered throughout the rest of the white matter. In the lumbar enlargement the degeneration was confined to the crossed pyramidal tracts, the posterior columns, and indeed all the rest of the cord was normal. The nerve cells throughout the cord contained large quantities of yellow pigment. The vessels of the white and gray matter showed marked hyaline degeneration, the walls being thickened and staining very diffusely. The roots reacted normally to all methods of staining except the Marchi. By it distinct degeneration was shown in the posterior, and questionable degeneration in the anterior roots. In the cord the sclerotic and degenerated areas contained a large number of black dots and compound granule cells.

To sum up: there was in the cervical and dorsal cord diffuse degeneration throughout the entire white matter, with dense sclerosis in the posterior and lateral columns; in the lumbar cord degeneration with sclerosis confined to the lateral columns; hyaline change in the blood vessels, and degeneration of the roots by the Marchi method. (See Fig. 1.)

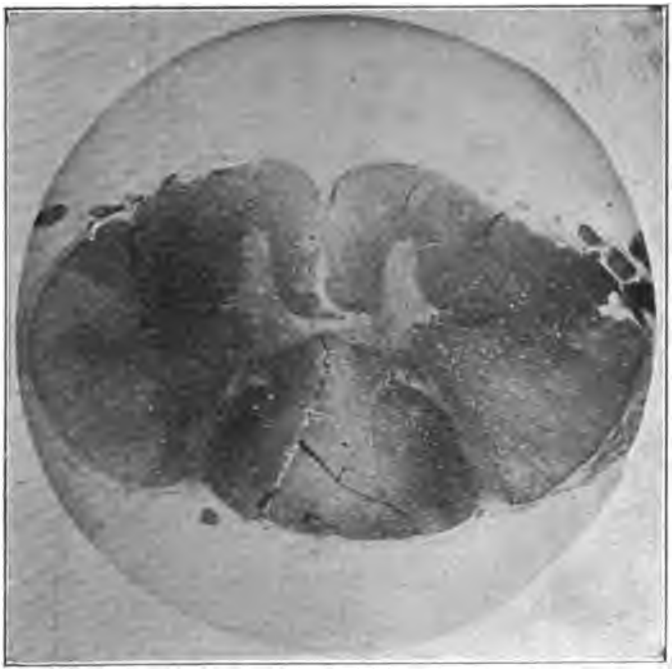

Fig. 1—Diffuse degeneration throughout the section with a secondary type of degeneration in the posterior columns.

The history of the third case, a typical example of pernicious anemia, is as follows: The patient, an insurance agent, fifty-six years old, was admitted to Dr. Morris J. Lewis' ward at the Infirmary for Nervous Diseases, complaining of weakness, and with manifest anemia. The family and personal history revealed nothing of importance. He admitted having had gonorrhea, but denied syphilis, and showed no signs of it. The illness for which he came to the hospital had begun two years before. The first symptom of which he complained was sore mouth and ulceration of the gums. This had lasted many months, but finally healed. In the meanwhile he had been gradually losing strength and he noticed numbness in the hands and feet. More recently pain in the dorsal and cervical spine appeared, and he began to have shortness of breath.

*Examination* revealed a sallow, poorly-nourished man. There was a little numbness in the arms and legs, but no true anesthesia. The ulnar and musculo-spiral nerves were tender to touch. There was great muscular weakness, but no true palsy. The urine contained neither albumin, sugar, nor casts.

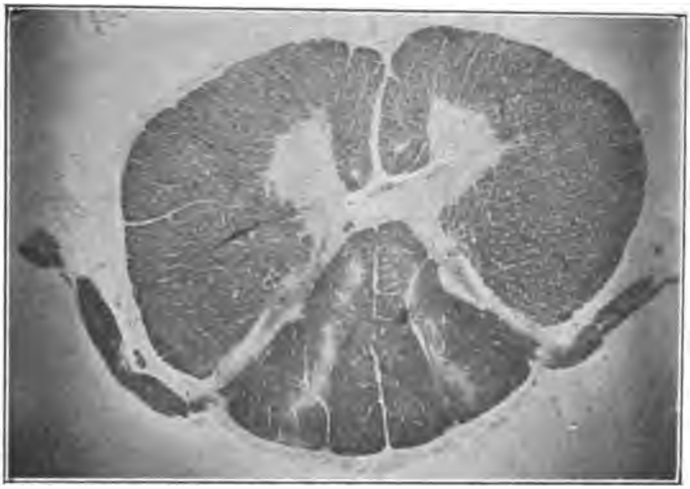

Fig. 2—Photograph showing diffuse degeneration in the posterior columns. This was present in only a small portion of the cord.

The lowest blood count showed hemoglobin 15 per cent.; red corpuscles 532,000; white corpuscles 1,500.

He died on November 23, 1901. The necropsy revealed the changes common in pernicious anemia: orange-colored, subcutaneous fat, red marrow in the long bones, atrophy of the gastric mucosa, etc.

The spinal cord changes consisted in a U-shaped area of degeneration in the posterior columns, each leg of the U being situated in the postero-external column and extending close to the margin of the cord. (See Fig. 2.) This area of degeneration extended from the fifth cervical to the second dorsal segment. There was very little sclerosis, the degenerated area consisting of a reticular tissue, with degenerated myelin, and, as shown by the Marchi method, fat globules and compound granule cells along the vessels. The vessel walls and ganglion cells were normal.

The fourth case is that of an English woman, thirty-four years old, who was admitted to the Philadelphia Hospital, January 30, 1901, complaining of loss of power in the legs. She had used alcohol moderately and tea to excess. Neither her history nor physical examination revealed any evidence of syphilis. A year before admission she began to have creeping feelings, and later distinct lancinating pains in the legs. A few months later she began to lose power in the legs, which increased until finally she was chair-ridden. The bladder and rectum were not affected at any time.

*Examination.* She was a thin, undersized, weak-looking, pale woman. There was marked loss of power in the legs. She was not able to stand up, indeed she could not lift either leg entirely off the bed. She could flex and extend the knee while the weight of the leg was supported by the heel upon the bed, could move the thigh weakly in any direction, and could flex and extend the ankles and toes. There was not only palsy, but also ataxia in the movements of the legs. The arms were weak but neither paralytic nor ataxic. There was no local muscular wasting, but marked general emaciation. Sensibility to touch and temperature was normal, but on the legs there was some impairment of sensibility to pain. The heart and lungs were normal. The urine contained neither albumin, sugar nor casts. The leg reflexes were absent. The first examination of the blood showed red corpuscles 894,000, white corpuscles 10,000, and 40 per cent. of hemoglobin. A second examination six weeks later showed red corpuscles 2,780,000; white corpuscles 8,000, and 43 per cent of hemoglobin. The further notes of the case are lost but we can with certainty of correctness state that under treatment with Blaud's pill, Fowler's solution, rest in bed, and feeding she improved so much that she put on weight, improved greatly in color, and walked without any difficulty. The red corpuscle count and percentage of hemoglobin rose quite high, but we cannot give the figures. She was discharged at her own request "feeling very well." The remaining cases were due to causes other than anemia.

Case five, J. P. G., fifty-nine years old, machinist, was admitted to the Philadelphia Hospital, April 24, 1901. He had used alcohol to excess for a long time, and had a chancre when 47 years old. About three years before admission his legs began to feel tired, heavy, and weak, and at the same time or soon after the stiffness, which made it difficult for him to rise, came on. One morning in August of 1900 he awoke and found his legs anesthetic from the hips down and so stiff that he could not move them. The next day he was able "to hobble around," and in a few days sensibility partially returned. There was for a time a distinct girdle sensation. For a few days before admission he had some urgency of micturition.

*Examination.* He was a man of spare build, but showed no local muscular wasting; indeed on account of the constant spasm the legs were proportionately better developed than the arms. Gait was very spastic. There was some true loss of power in the legs, none in the arms. He could not stand with the eyes closed. All the normal reflexes in the legs were exaggerated. The Babinski reflex was present on both sides. Ankle-clonus and rectus-clonus were marked. Rigidity of the legs from spasm was very marked. There was anesthesia to touch in irregularly scattered areas over the trunk and extremities, but sensibility to pain and temperature was normal. There was no ataxia in the arms. There was no nystagmus. Speech was normal. The urine continuously contained albumin and casts. He died of Bright's disease on June 1, 1901.

The spinal cord presented the following condition: There was degeneration which in the cervical region was differently placed in the upper and lower parts. The second and third cervical segments presented by the Weigert method very marked degeneration in the entire postero-median columns, and quite marked degeneration in the middle part of the postero-external columns. In the fifth, sixth and seventh cervical segments the disease continued distinct in the postero-median columns, but was much less noticeable in the postero-external columns, and also affected the direct and crossed pyramidal and direct cerebellar tracts. At these levels also there was some diffuse degeneration with a tendency to reticular formation throughout the entire white matter. Secondary degeneration had taken place, to any degree, however, only in the posterior and crossed pyramidal tracts. Both in the posterior and lateral and to a marked degree in the direct pyramidal tracts the rapid degeneration had led to a cribriform appearance of the tissue. In the dorsal cord the degeneration was of the same character as that of the cervical swelling, except that in the lateral and antero-lateral columns the diffuse degeneration was less distinct outside the direct and crossed pyramidal and direct cerebellar tracts. In the lumbar cord the degeneration in the posterior columns was confined to a triangular patch in each postero-median column, the bases of the triangles being parallel to each other along the posterior fissure. (See Fig. 3.)

The blood vessels of the pia and of the cord outside of the sclerotic areas were normal; within them there was slight thickening of the vessel walls and a webbed appearance along the course of the vessels, due probably to the presence of compound granule cells. The cells of the anterior horns were pigmented, but otherwise reacted normally to the Nissl stain. The Marchi method revealed a few compound granule cells, stained black along the course of the vessels in the sclerosed areas. The anterior and to a greater degree the posterior roots contained many black granules.

Case six, R. J. P., a sailor, sixty-nine years old, was admitted to the Philadelphia Home for Incurables, February 1, 1901. Four years before admission his legs began to feel tired and weak and stiff, and slowly grew worse, until finally he could scarcely walk and could stand only by balancing himself against the wall. Sexual power disappeared two years before admission and about the same time he became deaf in the right ear.

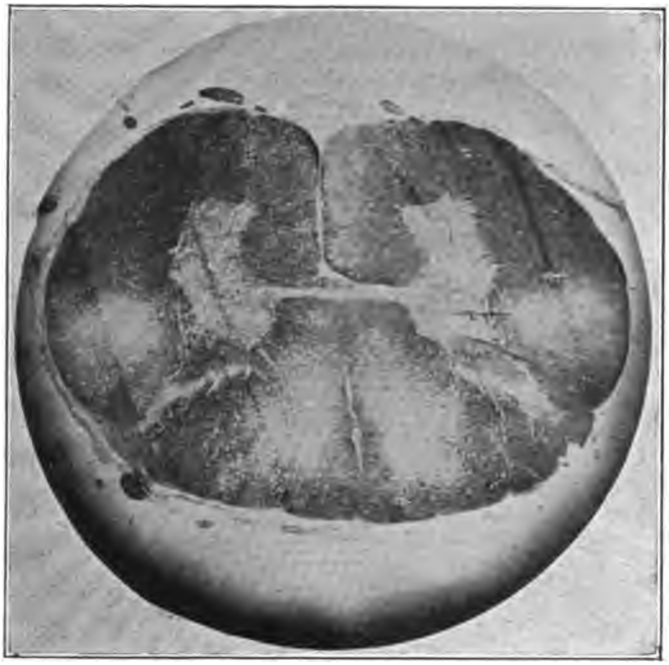

Fig. 3—Photograph showing a very diffuse type of degeneration with well marked reticular formation.

*Examination.* He was a well-built man with pigmented sores scattered over the trunk and extremities. There was no muscular atrophy. He had considerable difficulty in talking, his speech being a peculiar stammer with a decided tendency to scanning. [He said he had always had a slight impediment in speech, "but nothing compared to what it is now."] Static and motor ataxia was marked in the legs. Both legs were also spastic and weak. The knee, Achilles and cremasteric reflexes were exaggerated. The Babinski reflex was present on both sides. The biceps and triceps jerks were absent on the left side and very marked on the right. The right pupil, irregular in shape, reacted sluggishly to light, but with accommodation well. The irregular left pupil was rigidly fixed. There was anesthesia to all stimuli on both legs up to the knee and on the left thigh in a small area. The individual eye movements were good, but there was decided lateral nystagmus. There was a well-marked coarse intention tremor of the hands and head. Mental power degenerated during his stay in the Home, the tremor increased, speech became typically scanning; on January 22, 1902 he had a general convulsion with unconsciousness and he died three days later. The urine contained small quantities of albumin and granular casts.

Necropsy revealed the following: The convolutions over the anterior half of the brain were wasted, the pia adherent and somewhat thickened. There was much edema over the posterior part of the brain. The dura was normal. To the naked eye the spinal cord showed no lesion. When the brain was opened, after having been in a ten per cent. solution of formalin for twenty-four hours, more than a medium grade of internal hydrocephalus was present. The aqueduct of Sylvius and the fourth ventricle were also distended. The ependyma of the second and third ventricles was thickened, rough and easily separated as a leathery membrane from the subependymal tissue. The ependyma of the fourth ventricle was granular.

Microscopic examination: The cerebral cortex was somewhat atrophied. The cortical cells appeared normal in structure when treated by the ordinary nuclear stains. There was a low grade, productive meningitis of the pia-arachnoid. The vessels of the pia showed a well-defined endarteritis. The tangential fibers were at the best few and in some areas entirely absent. The ependyma was irregularly proliferated, there being heaps of cells in some places instead of a single row. Beneath the altered ependyma the glia was densely thickened and in it lay a plexus of vessels around which were several layers of small, round nucleated cells. As the thickened glia shaded off into the normal reticulum the vessels still retained their sheath of one or two rows of nuclei. The same condition existed in the lining of the fourth ventricle. Examination of the basal ganglia, the pons and medulla gave findings similar to those in the subependymal tissues of the cerebrum. There was a very marked gliosis beneath the ependyma of the fourth ventricle, and the ependymal cells there showed very marked proliferation, whirls of cells in some areas projecting into the lumen of the ventricle. Endarteritis and periarteritis were present in the pons and in less degree in the medulla. The pia-arachnoid of the medulla contained accumulations of small round cells here and there, but there was not the marked productive meningitis seen in the cortex. The nuclear ganglion cells of the twelfth and nucleus ambiguus were the seat of a diffuse chromatolysis. The pyramids were normal.

The meninges of the cord were normal. A slight degeneration shown by dropped out nerve fibers here and there was present in the lateral tracts and postero-median columns. A slight but distinct sclerosis was shown by the glial stains in these areas. The remainder of the cord was normal. The root zones in the dorsal and lumbar areas remained intact. The degeneration of the lateral columns in the lumbar enlargement was confined to the crossed pyramidal tracts. The nerve roots were not degenerated. The vessels of the cord itself showed no changes except that the media of the anterior spinal artery was perhaps a trifle thickened but not enough to have attracted attention had the cerebral vessels been normal.

Case seven, J. A., a laborer, fifty-five years old, was admitted to the Philadelphia Hospital, September 24, 1900. He had had syphilis some years before. He could not say how long he had been ill nor how his trouble began. He had been under the care of one of us (C.W.B.) three years before admission, and at that time had pain in the legs and back, a spastic and ataxic gait, increased reflexes in the legs, ankle-clonus, slight spasmodic retention of urine, no muscular wasting and normal pupillary reflexes.

*Examination.* There were weakness and spastic rigidity in both legs, more marked in the left. He could walk only by pushing a chair in front of him. There was some weakness of the left arm and left side of the face. He could not stand alone on account of both spasm and ataxia. There was some tremor of the tongue and slight ptosis of the left eyelid. He had some spasmodic retention of urine. Speech was slow but distinct. The normal reflexes of the legs were all much increased. The Babinski reflex was present. Ankle-clonus could not be obtained, probably because of the extreme spastic rigidity. The arm reflexes were absent. The pupillary reflexes were normal. He died on March 30, 1901.

The spinal cord lesions were as follows: The cervical cord stained by hematoxylin and by the Weigert method showed a diffuse sclerosis very dense in the posterior and lateral columns, and also a narrow band around the periphery and following the blood vessels inward. The nuclear stains showed a well developed productive meningitis. There was marked endarteritis of the vessels of the cord. The media were especially thickened. In short there was a specific meningomyelitis. Both the anterior and posterior roots were degenerated. The anterior horn cells were normal by the nuclear, carmine and Nissl stains. There was an ascending degeneration in the postero-median columns, and annular degeneration in the postero-external columns.

In the dorsal cord the degeneration was more marked than in either the cervical or lumbar. The posterior columns were affected in their entire area except in the root zones. The lateral tracts were also densely sclerosed, with the annular area of sclerosis in the antero-lateral and direct pyramidal tracts noted in the cervical region. The meningitis was more marked here than anywhere else in the cord.

In the lumbar cord the degeneration in the posterior columns was very slight and affected only the periphery. The roots were slightly degenerated. Besides the annular degeneration, there was also in the lateral column a wedge-shape area of degeneration of the crossed pyramidal tract, following the type of a descending degeneration from the dorsal lesion. The anterior horn cells were normal, but contained large quantities of the yellow pigment so commonly seen in these cells.

Case eight, J. J. M., a man fifty-three years old, was admitted to the Philadelphia Hospital, May 31, 1900. His memory was so poor and his answers were so contradictory that we could learn nothing about the history of his illness.

*Examination.* He was well nourished. The facial expression was dull, but except the poor memory he shôwed no other psychic symptoms than those common in stupid men. There was no muscular atrophy. The muscle power was weak. The knee-jerks were exaggerated, but ankle-clonus could not be obtained, probably because of the spastic condition of the muscles. The Babinski reflex was present on both sides. Sensibility especially for temperature was diminished on the legs. There was considerable ataxia and in the arm movements like a very coarse intention tremor. He had marked static ataxia and could not walk unaided. The symptoms remained unchanged until the death of the patient from cardiac failure and edema of the lungs on July 4, 1900.

The brain exhibited no gross changes. In the cervical enlargement sections treated by the Weigert sheath stain revealed a dense sclerosis affecting the entire posterior columns, the crossed pyramidal and the direct cerebellar tracts. The remainder of the cord was entirely normal. The degeneration in the posterior columns could be followed to the lower dorsal region where it gradually disappeared, growing less section by section. The degeneration in the crossed pyramidal tract was equally well marked throughout the entire length of the cord down to the lower lumbar region. The direct cerebellar tract was likewise degenerated throughout its whole course. In the posterior columns the root zone was much degenerated. In the postero-median column it was hard to find a trace of myelin, so dense was the sclerosis. The same was true of the greater portion of the postero-external column, although a few scattered myelinated fibers were seen near the gray matter. The sclerosis in the crossed pyramidal tract was almost as dense as that in the posterior columns. There were more stained fibers in the direct cerebellar tract than in either of the other two. The spinal roots showed no degeneration.

The Marchi method revealed very few degenerated fibers, but there were along the course of the vessels and more markedly in the lateral than in the posterior columns numerous compound granule cells which stained black. The roots, treated by this method, showed no degeneration. The degeneration could not be followed above the medulla; in the posterior columns it extended up to their nuclei; in the lateral tracts to the decussation. The cells of the anterior horns contained much yellow pigment, but were otherwise normal. The meninges showed an increased number of nuclei, but a distinct meningitis could not be said to exist. The vessels of the meninges and of the cord showed everywhere slight, and in the sclerosed areas marked, thickening of the media.

To sum up we had in this case a system degeneration affecting the crossed pyramidal tracts, the direct cerebellar tracts, and the posterior columns throughout their entire course, the nerve roots and the remainder of the cord reacting normally to all the different staining methods except that of Nissl.

Remarks: There are in the collection described above five cases of diffuse degeneration affecting the posterior and lateral columns, in four of which there was an associated intense anemia. In only one was the anemia pernicious in type, in another it was associated with senility, and in a third with parenchymatous nephritis. The histological changes in this group do not differ from those already described by Minnich, Nonne, Dana, Putnam, Burr and in the later contribution of Putnam and Taylor.

In cases one and six there was marked central gliosis. In case one, associated with anemia secondary to parenchymatous nephritis, the gliosis increased downward from the cervical region until in the lumbar cord it constituted a distinct tumor formation destroying the posterior commissure and explaining the loss of the pain sense. Subjective disturbances of sensation are common in the early stages of this group, and loss or impairment of the pain sense is more frequent than interference with the other forms of sensibility. The only constant lesion of the gray matter in this group, if indeed it be in truth a lesion, was intense pigmentation of the cells in the anterior horns. No distinct isolated hemorrhages were seen in any of the sections. We see nothing in these cases against accepting the intoxication theory to explain the diffuse degeneration and much in its favor. In the two cases in which nephritis and uremia were present this theory seems more rational than any other. The vessel changes were such as can be explained by the involvement of the vessel walls in the sclerotic process. There was, however, in the case of senile anemia a diffuse hyaline change independent of the sclerotic areas, and in the lumbar enlargement where the sclerosis was least, the degeneration of the vessel walls was as marked as in the most sclerotic parts of the cord. We do not attribute any special importance to the degeneration of the roots, seen only in the Marchi preparations, and do not consider it intense enough to explain the pains and disappearance of the reflexes. Though we have included these cases under the general title of postero-lateral scleroses, the process was not confined strictly to those tracts. The subacute degeneration affected in all our cases the lateral and anterior portions of the cord, but the secondary sclerotic process in the posterior and lateral columns was most intense in these areas, and the clinical phenomena are referable to them.

The two cases of syphilis of the nervous system illustrate two different types of disease due to the same cause. One (case seven) presented the lesions of a meningo-myelitis and a dense sclerosis of the posterior and lateral columns. Throughout the rest of the cord a slight perivascular sclerosis was present. There was also a ring of marginal sclerosis. The clinical picture differed but slightly from that of the long-standing cases of subacute degeneration.

The other (case six) is, to us, the most interesting of those here reported. In 1900 we reported to this Association[1] a case that showed at different periods the clinical picture of tabes, non-syphilitic postero-lateral sclerosis, and spastic paraplegia, and at necropsy presented the lesions of multiple sclerosis. We refer to that case here, and have included it in the series, in order to contrast it with case six which in life gave as typical a clinical picture of disseminated sclerosis as we have ever seen, and post-mortem an equally typical histologic picture of cerebro-spinal syphilis. The degeneration of the spinal cord was strictly confined to the posterior and lateral columns, without meningeal lesions and with such slight changes in the vessels that they can be disregarded in explaining the degeneration. There was also a very slight diffuse sclerosis of the posterior and lateral columns, following a system type, with sufficient defect in the myelin to give clear pictures to the naked eye after treatment by the sheath stains. This intrinsic degeneration of the cord and the lesions of the ependymal and subependymal tissues point to some intoxicating agent, as the lesions were identical with those described by us in a case of acute internal hydrocephalus for which no cause was found.[2] In investigating it we carried out a series of experiments on lower animals to study the effects of toxic materials placed within the ventricles. The reaction of the ependyma and underlying tissues in the intoxication experiments were strikingly like those in case six, and in the case of internal hydrocephalus. Tuberculin, sterilized urine, diphtheria toxin, dilute hydrochloric acid, and dilute carbolic acid all gave practically the same results, varying only in degree. The spinal cords of the animals were not studied.

Though the differential diagnosis between multiple sclerosis and multiple disseminated syphilis of the nervous system has been discussed by Gowers, Sachs and many others, this case and those reported by v. Bechterew and Krewer[3] lead us to agree with Oppenheim that a differential diagnosis must sometimes remain doubtful. Krewer's case showed at first a clinical picture of syphilis which later was replaced by a typical clinical picture of multiple sclerosis. There was no necropsy in it. In von Bechterew's case, as quoted by Krewer, the lesions resembled very closely those found in our case. There was, however, no hydrocephalus. That multiple sclerosis may present the clinical picture of a postero-lateral sclerosis is shown by the case reported by us.

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[1] JOURNAL OF NERVOUS AND MENTAL DISEASE, Dec., 1900.
[2] Journal of Experimental Medicine, Oct., 1900.
[3] Zeitschrift f. klin. Med., 1899.

The subject of combined system disease has been so thoroughly thrashed out without reaching any positive conclusion, that we shall not discuss the subject further than to state that case eight belongs to a group in which the degeneration is localized in certain fiber systems of the cord, usually the posterior columns, and the crossed pyramidal and direct cerebellar tracts. By the term combined system disease we do not mean to imply that the entire neurone to which the fiber belongs is affected, but only the portion in the cord damaged by the causative agent. We would differentiate this group from the subacute or diffuse degeneration group by the distinct limitation of the pathological process to the tracts above mentioned. We recognize fully that sometimes, when a local focus of softening has occurred, such as the area in the posterior columns in case three, should the patient live long enough a secondary degeneration might occur, and in so far the lesion be systemic. We think that such was the case in case one in which the patient so far recovered as to resume work and in which the degeneration and sclerosis in the posterior median columns were very dense, resembling to that extent sections from a case of dorso-lumbar tabes.

This small group of cases illustrates how difficult it is to arrange a classification of the diseases of the white matter in which the posterior and lateral columns are affected alone, or associated with minor changes in the other tracts, which with our present knowledge of the anatomy and physiology of the cord can be of no diagnostic value. For clinical and laboratory purposes we may arrange such cases as follows:

1. Friedreich's ataxia.

2. Tabes with associated diffuse sclerosis extending into the lateral columns. To this group may be added those cases of tabes associated with paretic dementia with secondary lesions in the crossed pyramidal tracts.

3. Tabes with degeneration in the crossed pyramidal and

also in the direct cerebellar tracts, with or without degeneration in Clarke's column.

4. Posterior sclerosis with sclerosis of the lateral columns and disease of the anterior horns. (Chronic poliomyelitis.)

5. Primary lateral sclerosis with minor changes in the posterior columns.

6. Subacute diffuse degeneration of the spinal cord due to anemia, cachexias, sepsis, *etc.*, with sclerosis in the posterior and lateral tracts predominating both in clinical manifestation and under the microscope.

7. Diffuse interstitial sclerosis, seen occasionally in chronic alcoholism with multiple neuritis,[4] in which the parenchymatous degeneration is secondary to the overgrowth of glial and connective tissue elements. To this same group on account of the similarity of the pathological process may be added the syphilitic postero-lateral scleroses secondary to meningeal lesions.

8. A combined system disease of unknown origin affecting the posterior and lateral columns, and distinctly confined to the direct and crossed pyramidal tracts and the posterior columns; the direct cerebellar tract may also be involved.

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[4] Phil. Med. Journal, Nov. 2, 1901.

# PARALYSIS OF ALL FOUR LIMBS AND OF ONE SIDE OF THE FACE, WITH DISSOCIATION OF SENSATION, DEVELOPING IN A FEW HOURS AND RESULTING FROM MENINGO-MYELOENCEPHALITIS[1].

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We are familiar with acute syphilitic meningo-myelitis chiefly as disease of the thoracic cord. Out of twelve cases tabulated by Williamson[2], in ten the lesion was wholly or almost wholly thoracic. In one it was thoracic and lumbar, in the other it was thoracic and cervical. The cases which have fallen under our observation have been thoracico-lumbar chiefly. It may be said that the localization to one portion of the cord is seldom absolute, in other words, although the lesion may be situated chiefly in one region of the cord, as the thoracic, some infiltration of round cells, and other evidences of meningo-myelitis are usually found in other regions. Cervical or cervico-bulbar forms of acute syphilitic meningo-myelitis, which might be designated clinically as high myelitis, are almost as rare as high tabes compared with the tabes which attacks the cord at lower levels, but in this cervical form the lesions are not confined to the cervical region, and are merely most intense in this portion of the spinal cord. Syphilitic meningo-myelitis is often associated with meningo-encephalitis. The following case is therefore in the first place of interest because of the unusual location of the myelitis. It also has other points of interest, as the manner in which the facial nerve and its nucleus on one side were attacked, its simulation of hemorrhage into the cervical portion of the spinal cord, and the interesting sensory and reflex phenomena.

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[1] Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

The patient, a colored man, forty-five years old, a laborer, was admitted to the Men's Nervous Wards of the Philadelphia Hospital, January 28, 1902. He had had gonorrhea twice, and a sore on the penis two years before. He had been a hard drinker. His family history was unimportant.

On September 26, 1901, the patient stated that he fell from a wagon, striking on his shoulder. He immediately rose, climbed to his wagon and continued working. Two weeks later he was obliged to quit work on account of pain in his left arm. He went to the Philadelphia Hospital for treatment, where he remained for about three weeks, at the end of which time he was again able to work. He worked until December 25, 1901, when pain in his left arm and between his shoulders compelled him to discontinue. The pain lasted for about one month.

At about 3 P.M., January 22, 1902, he suddenly lost the use of his left arm, this paralysis being followed in a short time by loss of control of the left leg; later by paralysis of the right side of the face, of the right arm and then of the right leg. Five or six hours elapsed between the time when he first noticed paralysis of the left arm and the time when the right leg became paralyzed.

He complained of swelling and of dull pain in the parts after they were paralyzed, but more especially in the left arm. He felt no pain immediately preceding the paralysis, and he was not unconscious at the time. He was not aphasic. After the paralysis he had a severe headache which ceased only the day before his admission to the hospital.

On admission (January 28, 1902), his general appearance was that of a well developed and well nourished colored man. He had had no movement of his bowels for five days before admission, and had voided no urine for at least twelve hours. Examination showed right facial paralysis, including the frontalis and orbicularis palpebrarum muscles. When the eyebrows were elevated no wrinkles were produced on the right side of the face. He was unable to fully close the right eye, and lachrymation was present. Speech was muffled, the mouth being drawn to the left side when attempts were made to talk, and whistling was impossible. Paralysis of the sixth nerve was not apparent. Hearing on the left side had been affected since the patient was eight or nine years old, and he could not hear the tick of a watch close to his ear; on the right he could hear the tick at about ten or twelve inches. Tinnitus aurium was not present. He had no difficulty in swallowing, but complained of difficulty in keeping food in his mouth when eating. The uvula was not deflected to either side. His sense of taste was preserved, a solution of quinine and spirits of camphor and sweetened water being recognized on the tip and right half of the tongue. The tongue deviated slightly to the left. The pupils were contracted, the left irregular in outline; irides reacted sluggishly to light and on accommodation. Hemianopsia was not apparent. Knee-jerks and Babinski reflex were absent; the grip on both the right and left side was zero.

His chest appeared normal, the clavicles were large and prominent, and the supraclavicular fossæ depressed. The lungs were resonant anteriorly. Examination of the heart revealed a systolic murmur; his pulse was rapid, full and easily compressed. His abdomen was normal in appearance and was not distended.

The patient was again examined on January twenty-ninth. His breathing was labored, and his voice weaker than on the day previous. He complained of pain in his shoulder. On the thirtieth of January his breathing was less labored. He did not talk much, and answered by nodding his head. It was reported that he passed blood with his stools. His appetite was good and he had slept well the night previous.

Between January twenty-ninth and February first he was visited several times by both of us. The results of our examinations are summarized below, his general condition not changing much during this period.

The patient was fully conscious, and had no aphasia. His voice was a mere whisper, but he understood all that was said to him. He said positively that he had not been unconscious even during the development of the paralysis, and he was equally positive that he had not had convulsions. Wrinkles had disappeared on the right side of his forehead. Bilateral ptosis, equal on both sides, was present, and could not be overcome by voluntary efforts to open his eyes. When the eyelids were closed distinct paresis of the right orbicularis palpebrarum was present, the right palpebral fissure being about 5 cm. in width. In showing his teeth his mouth was drawn to the left side, the right side of his face remaining immobile. The tongue protruded very slightly toward the left, even when the right corner of the mouth was drawn up by the hand of the examiner; the masseter contracted firmly; the uvula was in the median line, and no distinct paralysis of the soft palate was present. The pupils were equal; both irides responded to light and promptly in convergence. The eyeballs exhibited nothing abnormal. Movements of the head from side to side and from before backward could be freely made.

The patient could shrug his shoulders well, but with the exception of this movement paralysis of both upper extremities was complete. The limbs were flaccid, no contracture being present. Both lower limbs were paralyzed in all parts. The ophthalmic (supraorbital) reflex was present on the left, absent on the right. The conjunctival reflex was normal on the left, and greatly diminished on the right. Jaw-jerk was slightly exaggerated. The coraco-brachial or inner shoulder reflex was present. Biceps-jerk was almost lost on each side; the triceps-jerk was preserved on each side. The wrist-reflex was lost on each side. Knee-jerk was present on each side, but diminished, a little prompter on the right. The ankle-jerk was present on each side, and slightly exaggerated on the right. Ankle-clonus and the Babinski reflex were absent on each side.

The sensory conditions of this patient were of unusual interest. Tactile sense was preserved on both sides of the face, in the upper extremities and on both sides of the thorax and abdomen. Temperature sense (tested for heat and cold), and pain sense, were preserved on both sides of the face and neck, but were lost on the right side of the chest and abdomen, and diminished on the left side of the same. Temperature and pain senses were also lost in the distal portions of the right upper extremity. At a point about three inches above the elbow, hot, cold, and painful impressions began to be recognized. The pain and temperature senses were diminished in the left upper extremity. Sensation for touch was fully preserved in the left lower extremity, and somewhat diminished in the distal portion of the right lower extremity. Sensation for heat, cold and pain was almost completely lost in the left lower extremity, and completely lost in the right.

Respiration was entirely costal in type. The heart-beat was very rapid; a loud systolic murmur was present, loudest at the second intercostal space on the left side; the second sound of the heart was accentuated. The murmur was fully as distinct in the second intercostal space on the right side as in the corresponding space on the left. He had had no disturbance of bowels or bladder previous to his paralytic attack. When examined on January twenty-ninth and thirtieth he had incontinence of urine and feces; catheterization was necessary. He had had no dizziness nor vomiting at any time preceding the paralysis. He stated that his sight had been poor for three months. No ophthalmoscopic examination was made.

On February first he was somewhat duller, and early in the day did not seem to know where he was. On the next day his condition was much the same, but his pulse and respiration were more rapid, and he complained of pain between the shoulders. On the third of February the patient frequently protruded his tongue to moisten his lips, and complained of his mouth being dry. He was duller and weaker than on the previous day, and did not pay so much attention to questions. His respirations were shallow. He had not slept well the night before. He refused milk, but asked occasionally for water; his appetite was fairly good. On February 4, 1902, at 2.30 A.M., he died.

A summary of the most important coarse pathological appearances found on necropsy is as follows: Edema of the spinal cord in the upper cervical region, adhesion of the dura: chronic splenitis and perisplenitis, old fibrous splenic adhesions; calcified tuberculous nodules of the right lung. A detailed account of the examination of the brain is subjoined.

The calvarium was thick, measuring at the occipital end 1.25 cm. and at the frontal end 1 cm. The dura was tightly adherent to the calvarium and a small new growth was present directly in front of the pituitary fossa, not extending above the surface of the dura. This growth had two distinct parts. A small nodule was present in the middle cerebral fossa (left), and was soft to the touch.

The brain was somewhat edematous in the parietal and occipital lobes. There was no indication of hemorrhage in the brain or pons, and no hemorrhage in either lateral ventricle was apparent. The fluid in the lateral ventricles was not increased in amount.

No distinct abnormalities of the choroid plexus were observed. Horizontal section of the upper portion of the basal nuclei revealed no pathological condition. No gross lesions were found in the brain. The masses noted in the base of the skull did not extend into the bone itself, but lay within the dura.

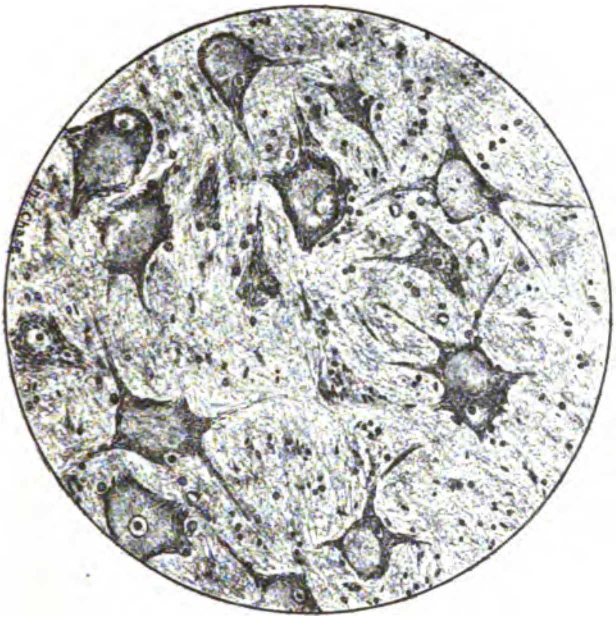
An increased amount of cerebrospinal fluid was found between the meninges and cord.

The brain and spinal cord were removed and examined microscopically.

Sections of the brain and spinal cord were stained by the Weigert hematoxylin and Marchi methods, by thionin, hemalum and acid fuchsin. Intense round-cell infiltration was found in the pia over the optic commissure, both in the walls of the vessels of the pia, and about some of the blood vessels within the commissure. The optic nerves and commissure as shown by Weigert's hematoxylin stain, were not degenerated.

Intense round-cell infiltration was found in the pia covering the medulla oblongata and the walls of some of the pial vessels were thickened. Here and there within the medulla oblongata a small vessel was found with round-cell infiltration about it.

Some of the cell-bodies of the left facial nucleus were degenerated, *i.e.,* the nucleus was displaced to the periphery of the cell-body and the chromophilic elements were broken up, but most of the cell-bodies of the left facial nucleus were normal or nearly normal. The right facial nucleus was exceedingly degenerated, and in most of the cell-bodies the chromophilic elements at the center of the cell-body had become granular or had disappeared, and only a few of these elements remained at the periphery of the cell-bodies; in others, no chromophilic elements at all were found. The nuclei were displaced to the periphery of the cell-body. The contrast afforded by the two facial nuclei was very striking. The intramedullary portion of the right facial root was deeply stained by the Weigert hematoxylin and did not appear to be degenerated by this method. The degeneration of the facial nerve was evidently of very recent origin, and it was impossible to say whether the disease of this nerve began in its nucleus or in its peripheral portion. The alteration of the cell-bodies was such as could be produced by disease of the peripheral portion of the nerve. The round-cell infiltration in the pia over the medulla oblongata was not greater on



Nerve cell-bodies of the right facial nucleus seen in one field of the microscope. The cell-bodies are drawn in their relative positions as shown by the microscope, and almost all are intensely degenerated.

the right side than on the left, so that the degeneration of the right facial nerve could hardly be explained by the meningitis. The nerve may have been injured in its peripheral portion by a small growth like those described within the dura of the base of the skull; and the paralysis may have existed longer than the patient was aware of.

The fourth, fifth and sixth cervical segments were so softened by inflammatory changes that the normal relations of white to gray matter were entirely altered, and a large portion of a transverse section at this level was not colored at all by the Weigert hematoxylin stain, because of degeneration of the medullary substance. The round-cell infiltration within the spinal cord and pia at this level was intense. Here and there were swollen axones or spaces from which the axones had disappeared. Some of the small blood vessels within the spinal cord and pia at this level had much thickened walls, and numerous very small hemorrhages were found within the spinal cord. Some of the vessels of the pia were almost entirely closed by the intense proliferation of the intima.

The round-cell infiltration within the pia was very intense at the eighth cervical segment, but considerably less so than at the area of softening. The veins of the pia were thickened, and in some of them round-cell infiltration was intense, especially within the inner coat. The small arteries of the pia at this level were not intensely thickened. No degeneration at the eighth cervical segment could be found by Weigert's hematoxylin method. The direct pyramidal tracts were found degenerated when the Marchi method was employed, but elsewhere at the eighth cervical segment the degeneration as shown by the Marchi method was very slight. Slight degeneration was found by this method in the left column of Burdach near the posterior horn, and may have been caused by degeneration of the posterior roots of this segment. The destruction of nerve fibers at the area of softening had not existed sufficiently long to cause much secondary degeneration detectable even by the Marchi method. Most of the cell-bodies of the lower cervical region, as shown by the thionin stain, were normal or nearly normal. Collections of small bacilli were found within the spinal cord at the eighth cervical segment. It seems hardly probable that these bacilli were the cause of the alteration of the central nervous system, and they may have invaded the spinal cord shortly before or shortly after death. The intense thickening of the blood vessels at the area of softening indicated that the process had been of long standing, and that the softening was probably the final result of a long-existing impaired circulation. The changes were such as are seen in syphilis, and the case probably was one of syphilis.

Round-cell infiltration was found in the pia of the mid-thoracic region, but was not quite so intense as in the eighth cervical segment. No degeneration by Weigert's hematoxylin stain was found at the mid-thoracic region.

The round-cell infiltration of the pia in the lumbar region was about as intense as in the mid-thoracic region. Some of the cell-bodies of the anterior horns of the lumbar region were tumefied and much pigmented.

In this case the rapidity with which the paralysis of the four extremities and of the face, with sensory, pulmonary and other symptoms of a serious character, developed made the diagnosis of spinal hemorrhage into the cervical region or into the oblongata-spinal transition seem probable, and this diagnosis was at first made. The pathological examination showed acute myelitis of high grade, the foundations of which were laid in previous disease of the vessels and membranes. Numerous small hemorrhages were present in the more or less inflamed and disintegrated cervical cord, but these were secondary, or at the most concomitant with the attack of myelitis. In severe acute myelitis hemorrhages almost invariably occur, these in some instances being punctiform, in others of considerable extent, even sufficient to cause some bulging of the cord; not infrequently both punctiform hemorrhages and those of larger size are present in the same case. The fundamental disease in these cases is, however, the myelitis, although this usually occurs on the basis of previous disease of the spinal vessels.

When the question of diagnosis is between myelitis and hemorrhage, this is understood to mean between primary hemorrhage into the spinal cord (hematomyelia) and myelitis, or between primary hemorrhage into the spinal membranes (hematorrhachis) and myelitis. The manner of onset may give the clue to the diagnosis between hemorrhage into the cord and acute myelitis. In hemorrhage the attack is usually sudden and without premonitory symptoms, although this rule is not without rare exceptions, as when a paralysis preceded by some paresthesia comes on slowly. The development of paralysis in the case here recorded was relatively rapid; nevertheless five hours were occupied in its completion. Prodromal symptoms were not entirely absent, even presuming that the pain in the arm which followed the fall of four months previous had no connection with the attack of myelitis, it will be remembered that for one month previous to the development of paralysis in the four extremities the man had had pain between his shoulders and in his left arm. Other prodromal symptoms were absent, but this was sufficient to cast some doubt upon the diagnosis of primary hemorrhage into the cord. The usual causes of hemorrhage were absent, unless the fall of four months previous may have been thought to bear some causal relation to the attack, but the traumatism was too remote to have induced a gross spinal hemorrhage. It was more likely to play a rôle in the determination of an inflammatory process in the spinal cord of a syphilitic subject. The patient had suffered from syphilis, the common cause of acute myelitis.

The diagnosis from spinal meningeal hemorrhage, extradural or subdural, was less difficult than that from primary hematomyelia. In spinal meningeal hemorrhage the symptoms of irritation come on suddenly and are usually of a very severe type, although even this rule is not without exceptions, probably in cases in which the hemorrhage into the spinal spaces occurs gradually. A meningeal hemorrhage sufficient to have produced the profound paralytic, sensory, reflex and other symptoms which were present in our case, would in all probability have been ushered in by severe pain in the back and in the distribution of the nerves whose roots were irritated by hemorrhage, and in addition by rigidity of the back, possibly local spasm in nerve distributions, contractures and even more or less generalized convulsions. With the exception of a steady pain in the back and one arm these irritative phenomena were not exhibited by the patient prior to the paralysis. In spinal meningeal hemorrhage paralysis usually occurs after the phenomena of irritation have been well marked, while in both acute myelitis and hematomyelia, whether primary or secondary, the paralysis is one of the earliest symptoms. The usual causes of spinal meningeal hemorrhage were absent in our case, while as already stated, syphilis, the common cause of myelitis, acute or chronic, was present. The course taken by the disease was not that of spinal meningeal hemorrhage, which usually results in death because of the extent of the inundation, or in partial recovery through absorption after the lapse of considerable time. The rapid development of the paralysis of all the limbs resulting from myelitis makes the case one of unusual interest.

Another disease from which a case like the one here recorded might need to be differentiated is spinal or cerebrospinal tuberculous meningomyelitis. A pure spinal tuberculous meningitis is rare, but the cerebrospinal affection is not uncommon, and when the lesions are basal and spinal, the latter predominating, the case may show some features similar to those presented by a syphilitic cervical or cervicothoracic myelitis of the acute or at least of the subacute type. A case has been reported by Hensen[8] in which the diagnosis of cerebrospinal syphilis was made, but in which the autopsy showed the case to be one primarily of tuberculous meningitis, inflammation and degeneration of the spinal cord taking place secondarily. This case is in itself one of unusual interest, and is worthy of being recorded in detail in this connection.

A woman forty years old became sick eight days before she was received into the hospital with chill and severe headache. These symptoms were soon followed by severe pain in the back and the region of the kidneys, especially on the right side. A few days later disturbances in micturition occurred, requiring catheterization. On the eighth day of her disease she was taken into the hospital with a diagnosis of kidney and bladder disease. On admission she was mentally disturbed, confused and excited. She had pains in her head and back, in the region of the kidneys and in the legs. The atlanto-occipital articulation was tender to pressure, but no rigidity of the neck was present. There was, however, extraordinary rigidity of the whole vertebral column. No vertebra, however, was especially tender. Slight abducens paresis on the right side; bladder distended, detrusor paralysis; some hyperesthesia in the legs; patellar reflex exaggerated; other reflexes normal. The patient with support and even alone was able to take a few steps, although with rigid back and legs. No choked disc, not even later. The statement that out of 12 children she had borne most of them had died in early life aroused the suspicion of syphilis, although no other symptoms of syphilis were obtained. Some improvement occurred in the patient's condition, the mental excitement diminished, and voluntary urination became possible. Temperature was diminished. On the other hand, however, rigidity of the neck and of the vertebral column became constant. The hyperesthesia also diminished. On the eighteenth day of the disease indisputable symptoms of spinal cord disease in the thoracic region developed. She complained of paresthesia in the right leg and then in the left. She was unable to raise the lower limbs. The muscles became flaccid, and marked reflex contractions developed in testing for the patellar reflex and in irritation of the skin. A few days later girdle pain in the abdomen developed on each side as high as the eighth rib; the abdominal reflex was lost. On the twentieth day of the disease the legs became completely paralyzed, and sensation in the lower parts of the body was almost lost. The patellar reflex was lost, the muscle excitability in the peroneal muscles on both right and left side was increased. In the left anterior tibial muscle there was beginning reaction of degeneration. Incontinence of feces and tendency to decubitus. Now cerebral symptoms again became conspicuous. The sensorium became clouded. The patient became somewhat delirious at night, and there was some evidence of facial paralysis which sometimes seemed stronger on the right side, sometimes stronger on the left. The abducens paralysis which at first had come on had disappeared. The muscles of the eye were not implicated. On the twenty-first and twenty-second days the mental disturbance developed into profound stupor, and slow forced movements appeared in the upper limbs, and only occasionally seemed to be somewhat coördinated. The diagnosis was made of cerebrospinal syphilis.

At the necropsy the diagnosis of syphilis was not confirmed, but a basal tuberculous meningitis with more extensive tuberculous meningitis of the spinal membranes, especially in the thoracic region, and secondary softening of the cord were found. Also miliary tuberculosis in the lungs, liver, kidneys and ulcers in the vermiform appendix.

The microscopical examination showed that the pia-arachnoid was in a state of inflammation throughout the entire length of the spinal cord, but more especially in the thoracic region. The inflammatory condition was more pronounced on the posterior aspect than on the anterior. The walls of the vessels, arteries as well as veins, were thickly infiltrated by round cells, almost difficult to recognize, and their layers could not be distinguished. Some vessels were thrombosed. The small-cell infiltration extended from the vessels into the surrounding pia. Distinct tubercles were not found, possibly on account of the short duration of the disease, but the absence of tubercles is not uncommon in tuberculosis of the cerebral membranes. However a few typical giant cells were present. In the middle thoracic region only scattered portions of the gray matter could be recognized, in which a few ganglion cells could be discovered after considerable searching. By the Pal stain a few normal nerve fibers were found, most of them, however, had disappeared. No tubercles were found in any part of the spinal cord substance.

From the pathologico-anatomical point of view the degeneration of the spinal cord was secondary to the meningeal disease, especially of the vessels of the meninges—everywhere where there was meningeal disease of high grade there was degeneration in the border zone of the spinal cord also, whereas in the thoracic region there was complete destruction and dissolution of the spinal cord substance. The primary lesion, however, was the vascular lesion of the meninges.

A comparative study of Hensen's case and of the case here recorded shows that in the case of tuberculous meningo-myelitis the paralytic, sensory and reflex symptoms of grave type developed gradually and after a period in which the symptoms of meningeal irritation were prominent. It was eighteen days before indisputable symptoms of involvement of the cord were exhibited, and twenty days before paralysis was complete. The extensive meningeal inflammation was shown by the rigidity of the back, and also probably by the hyperesthesia of the extremities. In our case paralysis of all four limbs was complete in five hours, and the grave sensory, reflex and visceral phenomena came on with almost equal rapidity. With the exception of pain between the shoulders and in the left arm, symptoms of meningeal inflammation were absent. Basal symptoms were present in both cases, but most of these developed in the tuberculous case late in the disease, while the facial paralysis in our case was a prominent feature in the paralytic syndrome which came to the surface in the short period of five hours if the patient's statement may be relied upon.

It is well to add that the diagnosis of tuberculous meningomyelitis in Hensen's case was made from the condition of the lungs and intestines, and not from the presence of tubercle bacilli within the spinal cord.[4]

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[2] "Syphilitic Diseases of the Spinal Cord," Manchester, 1899.

[3] Hensen, H., Deutsche Zeitschr. f. Nervenheilk., v. 21, No. 3-4, p. 240.

[4] A case of tuberculous meningomyelitis with paralysis has been published by Joseph Collins in the December, 1902, number of this JOURNAL.

# PHILADELPHIA NEUROLOGICAL SOCIETY.

October 28, 1902.

The President, Dr. John K. Mitchell, in the chair.

*Astasia-Abasia.*—Dr. F. X. Dercum referred to a case which he had intended to present to the Society, but the patient had recovered. He was a colored boy, thirteen years old, and had been struck on the back with a ball. This was followed in the course of a month by great incoördination in the legs, and for seven months he was incapacitated for walking. He could move the legs well while in bed on his back. He made a perfect recovery under suggestion, without hypnotism, and the use of a placebo.

*Peculiar Swelling of the Back of the Hand.*—Dr. F. X. Dercum exhibited a woman who had a negative family history, was married, and the mother of two healthy children, and had had no miscarriages. Twelve years ago she had a prolonged illness of a doubtful nature. During convalescence she had swelling of both feet and hands. This swelling disappeared except in the right hand, where it still persists. The swelling has undergone no change. Her weight increased remarkably six months ago, and she gained one hundred pounds in the course of a year.

The dorsal aspect of the hand presents a soft gelatinoid swelling like that of myxedema. The finger nails are ridged and brittle, and somewhat atrophic. She has pain about the roots of the nails and the skin of the distal phalanges is somewhat shiny.

Dr. T. Diller reported a case of myasthenia gravis pseudoparalytica.

Dr. C. K. Mills reported a case with symptoms of myasthenia gravis, resulting from an electric shock of 1,300 volts.

Dr. S. W. Morton and Dr. C. K. Mills reported a case of acute bulbospinal paralysis.

Dr. T. H. Weisenburg read a paper on the effects of electricity on the human body.

*Bulbar Palsy.*—Dr. W. G. Spiller presented a man, aged thirty-two years, who in September, 1901, first noticed a sensation of fatigue in the muscles of the lower part of the face. He had been drinking previously. The sense of fatigue usually was not caused by eating or by cold, but by talking or reading aloud, and was worse in the morning. He complained also of tremor in the lips and neck on the left side, and fibrillary tremors in the left side of the lower part of the face had once been observed in the clinic. The man was unable to elevate either corner of the mouth, and when he laughed the corners of the mouth were drawn outward, but very slightly upward. Sensation on both sides of the face was normal. The reactions of the muscles of the face to the faradic current were normal. Dr. Spiller presented the case as one of bulbar palsy or myasthenia gravis pseudoparalytica in the early stages.

Dr. Wharton Sinkler remarked that the case reported by Dr. Diller was extremely typical, although the author had not laid stress on one of the most characteristic features of these cases, that is, the extreme exhaustion of the muscles after exertion. He spoke of the speech being worse after talking, but did not refer to the effect of muscular exercise. An interesting point in connection with the case was the occurrence of angioneurotic edema. This Dr. Sinkler considered favored the view advanced by some writers that myasthenia gravis is due to an infection.

With regard to the case reported by Dr. Mills, he said that it did not look to him like a case of myasthenia gravis, but more like one of true bulbar paralysis, from the fact that there was atrophy of the tongue, facial muscles, and of the upper extremity, with exaggerated knee-jerks.

Dr. D. J. McCarthy said that in the examination of the nervous system made in a case of myasthenia gravis of Dr. Burr, the findings were negative, but in that case and in another with autopsy since, distinct lesions in the thymus gland had been found. The first case developed after pregnancy, and there was a persistent thymus gland as large as the fist, and the lymphoid tissue throughout the body was increased in amount. Examination of the muscles gave negative results. In the second case, examination of the thymus showed enlargement with an abscess as large as a small walnut in the central part. Examination of the nervous system and of the muscles gave negative results. He referred to other reported cases in which similar lesions of the thymus gland had been found.

Dr. S. W. Morton said that the only reason for mentioning electricity as a probable cause in his case was that careful study had failed to reveal anything else as a cause. In the case of bulbo-spinal paralysis presented by Dr. Mills, the same cause suggested itself. It seemed possible that the frequent application of weak currents might exhaust the nervous system. Many of these cases of acute paralysis are undoubtedly due to auto-intoxication, or to some form of poisoning. With this idea in mind, he had looked for enlargement of the spleen, but found none. He mentioned that the pharyngeal reflex was impaired, but not lost.

Dr. Alfred Gordon read a paper on the condition of sensation in motor paralysis of cerebral origin, and reported the results obtained by a study of thirty-five cases.

Dr. F. X. Dercum remarked that the most common condition referred to by Dr. Gordon was hypesthesia. That hypesthesia is very frequently present in hemiplegia is a matter of common experience. This is especially true in old cases. A possible cause of the hypesthesia in these cases is deficient nutrition in the paralyzed limbs, which are cold and livid and the seat of arrested functional activity. Under such conditions we should look for lesion of the nerve endings and diminished conductivity of the nerve trunks.

Dr. Dercum did not think that anesthesia was necessarily in proportion to the motor loss.

Dr. Charles K. Mills said that while Dr. Gordon's paper was an interesting contribution to the question, it did not seem to him that, granting the accuracy of the observations, the conclusions apparently drawn were entirely warranted. Of the thirty-five cases examined, there were a number which failed to show any of the sensory disturbances referred to, and the author had made no attempt to explain the character of the lesion in those cases in which the symptoms were essentially motor. The speaker also called attention to the fact that no attempt had been made to separate the different varieties of astereognosis.

The majority of the cases of hemiplegia seen at the Philadelphia Hospital are due to subcortical lesions. Many are due to capsular, thalamo-capsular, lenticulo-capsular, and lesions of considerable extent, often hemorrhagic, which have acted in different directions, and there is every reason to believe that they involve the separate tracts which exist in the corona radiata, and in the capsule, and in other parts.

While not agreeing in the conclusions of the author, he held that this form of clinical work was of the greatest importance in the solution of these problems, especially when confirmed by autopsy.

Dr. F. Savary Pearce said that it had occurred to his mind that the anesthesia, the hypesthesia, and the other sensory changes occurring early in hemiplegia might be accounted for by the vasomotor paresis together with the formation of toxic products, and their retention in the tissue of the paralysed side, thus affecting the tactile corpuscles and the peripheral part of the nervous system. This part after a time partly or in whole recovers. In the chronic cases the sensory disturbances are probably the result of lack of nutrition, as suggested by Dr. Dercum, and not necessarily due to changes in the central nervous system. He believed that in many cases the lesion is in the peripheral parts rather than in the brain itself.

Dr. William G. Spiller said that the paper was purely clinical, and that we should not attempt to draw anatomical conclusions from it. In two of the cases in his own ward, which Dr. Gordon had employed in his study, Dr. Spiller obtained necropsies. In one there was an area of sclerosis in the occipital lobe, extending forward and involving the posterior limb of the internal capsule. In the other the optic thalamus was implicated.

In connection with Dr. Gordon's paper, Dr. Spiller referred to the recent paper by Marie and Guillain, the latter paper being anatomical. These authors had shown how difficult it is to determine the cause of hemianesthesia in hemiplegia, from the lesions found.

Dr. D. J. McCarthy referred to a case, the specimens from which he was at present studying. During life there had been a quadraplegia; no loss of sensation, although muscular sense was not studied. The autopsy showed hemorrhage affecting the lemniscus and a small area about it. The secondary degeneration extended upward, affecting the posterior part of the internal capsule. There was also some degeneration of the cortex, but this had not as yet been sufficiently studied, to permit an exact determination of the area.

Dr. Alfred Gordon said that it had not been his intention to solve the question of localization. He had wished simply to approach the subject from one side, and to study in an impartial manner the question whether in these cases of motor paralysis of cerebral origin, there were sensory disturbances or not. The literature shows such variations in the results that he wished to study the matter for himself. The question of separate localization is still far from being solved. As Marie has suggested, why should we not, as the evidence is so contradictory, cease fixing our minds on the *carrefour sensitif,* and look for sensory fibers in some other part; just as with the motor fibers, we remove a portion of the motor cortex and the patient recovers with complete return of power; this shows that the motor area is not the only portion of the brain which controls the motor nerves.

Dr. F. X. Dercum reported a case of adiposis dolorosa with involvement of the joints.

# Periscope.

## NOUVELLE ICONOGRAPHIE DE LA SALPETRIERE.

(Vol. 15, 1902, No. 5, Sept.-Oct.)

1. The Deviation of the Vertebral Column in Parkinson's Disease. J. A. SICARD and L. ALQUIER.
2. The Syndrome of Spasmodic Torticollis. DESTARAC.
3. Study of the Root and Cell Lesions of Tabes. A. THOMAS and G. HAUSER.
4. Contribution to the Study of the Physiological Action of Some Bromides. C. FÉRÉ.
5. Does a Peroneal Form of Charcot Marie Amyotrophy Exist? P. SAINTON.

1. *Parkinson's Disease and the Vertebral Column.*—The authors of this article in observing cases of Parkinson's disease were impressed by the fact that the deviation of the vertebral column was a frequent occurrence among them. This point had not received any mention either in the special monographs on this subject or in the text-books. In seventeen cases of Parkinson, twelve presented definite deviations of the vertebral column. This large proportion does not seem to depend upon accidental circumstances, because in examining some old published cases and photographs, the authors found evidences of this same condition, which had escaped the attention of former writers. The variety of deviation was found to be distributed as follows: four kyphoses, one scoliosis, four kypho-scolioses, two lordo-scolioses, and six kypho-lordo-scolioses. This vertebral column affection cannot be dependent upon cachexia or the prolonged stay in bed, because three only of this series would come into such a category. The examination of these cases shows very plainly that the deviations are directly caused by muscular rigidity. This theory is noted by the authors in concluding the article, which is illustrated very well by photographs that emphasize the authors' standpoint.

2. *Spasmodic Torticollis.*—Destarac presented to the last Congress of Toulouse two patients showing the same group of symptoms which recalled on the one hand spasmodic paraplegia and on the other Friedreich's disease and hereditary cerebellar ataxia. In this latter affection the integrity of the muscular force is in sharp contrast to its functional impotence systematized under the form of functional spasm, as is shown in certain complicated and co-ordinated movements, such as walking and writing. A spasmodic torticollis seems to be the most striking phenomenon of this condition and for that reason the syndrome of spasmodic torticollis has been the term used to describe it. The author attempts to prove that such cases and Friedreich's disease and hereditary ataxia have a common symptomatology. The first case, a girl of eighteen, offers the following points of similarity to these two conditions: Its appearance in the eighth year of life. Difficulty in writing preceding all other symptoms by a long time. Peculiar position of the feet in walking. Involuntary movements of the head and trunk. Flat foot and scoliosis. Tachycardia, dyspnea and vertigo. Conservation of muscular power. The reflexes are increased and not absent as is most commonly found in Friedreich's. The second case shows even more similarity. Man twenty-nine years old; at the age of twelve, difficulty in writing. Abolition of tendon reflexes, with conservation of skin reflexes.

Flat foot, scoliosis. Static ataxia of right arm. Involuntary movements of the face. Speech defect. Fibrillary contraction. Slight decrease in objective sensibility, and the abolition of electro-muscular sensibility. Attacks of nystagmus, tachycardia and oppression. Modification of mechanical and electrical excitability, which recalls the myotonic reaction of Thomsen's disease. The cerebellar syndrome dominates the clinical picture of Friedreich's and hereditary ataxia, and the cerebellum appears to be either directly or indirectly most in evidence as a possible causative factor. After closely analyzing the symptoms present in these two cases and comparing them with those found in the above named diseases, Destarac concludes that they depend upon a disturbance of muscular tonus and of muscular equilibrium which manifests itself only under certain conditions, sometimes when the patient is in an upright position, and at other times when he attempts to execute certain complex movements with the conservation of muscular force. The many theories in regard to muscular tone may be summarized as follows: (1) Tone is the external manifestation of a state of permanent excitability of the motor cells of the cord, kept up by peripheral impressions. (2) This state of excitation of the motor cells is subject to a double influence, inhibitory or excitatory, arising from the superior centers (brain, cerebellum, medulla). These hypotheses applied to the cases in point, strongly support the conclusion that the relation between them and Friedreich's ataxia and hereditary ataxia is a very close one.

3. *Root and Cell Lesions of Tabes.*—This is a valuable contribution to the knowledge of the pathology of tabes which extends through two numbers of the journal. The article opens with a short consideration of the various theories which have been advanced to explain the origin of the tabetic lesion. The one point that stands out most definitely among all theories is the rôle which the posterior nerve roots play. It is the atrophy of these which occupies the preponderant position in all the results noted from the study of tabes. In spite of the recent work of Obersteiner, Redlich, and Nageotte, no definite decision has as yet been reached in respect to the cause, nature, and pathogenesis. For this reason the authors have made use of their large material to study the posterior nerve roots, their course between and in the neighborhood of the ganglion, above and below it. The ganglion itself and the alterations in the nerve cells have been investigated also. In the second portion of the article a very excellent anatomical description of the ganglion and its nerves is given, illustrated by very clear diagrams. The stains used were Weigert and Pal, with Azoulay for the nerve fibers and Marchi. As the specimens were mainly hardened in Müller the Nissl stain was seldom used. Forel's method of staining *en masse* was used with good effect for the axis cylinder, with an after treatment by Marchi. The third portion of the article is given to the description of the findings in the eleven cases of tabes which form the basis of this study. A short clinical résumé precedes each case. The description of the microscopical findings is very accurate and painstaking, and the illustrations are unusually good and convincing. The fourth portion of the paper is given up to a recapitulation of the findings, and the fifth to an interpretation of them. Under the former are noted (1) Lesions of the root fibers, the study of the root atrophy in the subganglionic tissue. (2) Lesions in the connective tissue of the ganglion. In all cases a pachymeningitis more or less pronounced existed about the posterior root. (3) Lesions in the parenchyma of the ganglion. The article ends with the authors' interpretation of these findings. In respect to the rôle which the nerve cells of the posterior root ganglia play in the origin of tabes, the following are noted: (1) The cellular lesions are inconstant, and the more recent the tabes, the less chance there is of finding them. (2) They are of little consequence in proportion to the number of cells present. (3) The changes are not speci-

fic in character; sometimes there is an alteration of protoplasm (chromatolysis, vacuolization, and atrophy) or of the nucleus (atrophy, disappearance); sometimes an inflammation of the pericellular capsule with hyperplasia of the cells or of the connective tissue. The following conclusions are noted as the results of this very interesting study:

The essential lesion of tabes is after all a dystrophy, which occurs in the whole of the peripheral sensory neurone, predominating especially in the central prolongation of the cell. This dystrophy attacks generally certain portions of the motor protoneurone and of the sympathetic system. The histological alterations are characterized by modifications in the nerve fibers which can be compared under certain conditions to those which occur in the course of a toxic neuritis, either produced experimentally or in the course of disease. They are very distinct from a Wallerian degeneration. If the cell body of the sensitive protoneurone appears in general to keep its structure and its normal aspect, it is nevertheless sometimes the seat of a trophic lesion. In addition, in spite of the absence of atomic lesions, it is reasonable to suppose that its trophic function is in a certain measure compromised.

4. *Bromide Action.*—This paper is a continuation of the experiments made by Féré to determine the effects of doses of bromide upon the work capacity. The cortex of the brain gives rise to voluntary muscular activity upon excitation, and the ergograph of Mosso is the means used to measure this. The mass of experimental data makes an abstract of this article an impossibility.

5. *Does a Peroneal Form of Charcot Marie Amyotrophy Exist?*—This question is answered in the affirmative by Sainton in a brief communication in which he mentions two cases of amyotrophy limited to the lower limbs. The grandmother of these individuals had the same condition which was limited as in them to the lower extremities. Although there has been no autopsy on such cases, the existence of a peroneal amyotrophy of the Charcot-Marie type cannot be doubted.

Sidney I. Schwab (St. Louis).

### AMERICAN JOURNAL OF INSANITY.

(Vol. LVIII, 1902, No. 4.)

1. Etiology of Paresis. Hurd.
2. The Early Diagnosis of Paresis. F. X. Dercum.
3. The Comparative Frequency of General Paresis. O. G. Wagner.
4. Treatment of Paresis; Its Limitations and Expectations. E. Cowles.
5. Heredity—With a Study of the Statistics of the New York State Hospitals. W. G. Krauss.
6. Senility and Senile Dementia. Russell.
7. Some Observations upon the Elimination of Indican, Acetone and Diacetic Acid in Various Psychoses. Coriat.
8. Studies in the Manic-Depressive Insanity, with Report of Autopsies in Two Cases. S. Paton.

1. *Etiology of Paresis.*—The author takes up the various factors bearing on the etiology of this disease, beginning with syphilis, citing the results of observations of different writers, which vary all the way from 11 to 94 per cent., while in his own cases he has found only 20 per cent. of paretics syphilitic, which he accounts for largely by the inability to secure a complete anamnesis in many cases of the disease. As a further argument in favor of lues, he shows that this disease is particularly rare where syphilis does not exist, as in Ireland; but again in Egypt and Japan, where it is extremely prevalent, these countries lack the special stress coincident with Western civilization. Further clergy, priests and Quakers, who are rarely syphilitic, are proportionately free from paresis. It has also been observed,

that paresis is much less prevalent in rural districts, where lues and mental stress are rare. He calls attention to the like relation of syphilis and paresis in men and women, *i.e.*, about 4 to 1. In juvenile cases hereditary syphilis has been found to the extent of 100 per cent. He ascribes little weight to heredity and considers paresis one of the least transmissible of mental diseases. He regards alcohol more as an incidental factor, and inebriety is often developed after the onset of the disease and therefore more a symptom than a cause. With respect to head injuries, they have no special etiological bearing and the effects of lead, tobacco, *etc.*, he regards as problematical. He believes the increased mental anxiety and over-exertion of the century just past is largely responsible for the startling increase in the frequency of paresis. The similarity of tabes and paresis has been urged in favor of the syphilitic etiology of paresis, for in the former disease a history of luetic infection is found in 80 to 90 per cent. of the cases. The failure of anti-specific treatment in paresis is ascribed to the different tissues affected in brain syphilis and paresis, in the latter the neurone, in the former the meninges, and the latter is incapable of regeneration. He concludes his paper: "That syphilis is the most common factor in the production of paresis. That it may cause it directly—an exciting cause. That it may cause it indirectly by bringing about such a devitalization of the system generally as to render other influences operative—a predisposing cause. That it is not usually the sole cause, but that there is associated with it the deleterious effect of mental stress and over-excitement, dissipation and alcoholism, and heredity. That in a certain relatively small number of cases mental stress, worry or overwork may be the sole ascertainable cause. That traumatism may also be the cause in a still smaller proportion of cases, but that in many of them it acts as a developing or ripening agent of an incipient paresis in a syphilitic subject."

2. *The Early Diagnosis of Paresis.*—Differentiation from neurasthenia difficult in the early stage, yet the symptoms only superficially similar, those of the neurasthenic being more subjective, while the paretic rarely complains or consults a physician, his friends narrate his symptoms. In neurasthenia the physical appearance rarely changed, or if so, differently from paresis, where the facial expression is changed in quality. The neurasthenic's movements are not altered in precision, not incoördinate. The neurasthenic's sleep disorders nearly the opposite of the paretic's; the neurasthenic is at his best in the evening, the opposite with the paretic, for then his little delinquencies in the matters of dress, deportment and speech defects become more prominent. Quantitative mental changes occur early in paresis, not so in neurasthenia; the neurasthenic is distinctly nosophobic, has no essential memory defect, still mental functions quickly tire. In paresis early mental fatigue, but loss of ability to remember and to comprehend is added; soon marked impairment of memory, manifested by neglect of attire, business, various improprieties, *etc.*, which are foreign to neurasthenia. The loose conception of neurasthenia largely chargeable for any confusion with paresis. This early stage he terms the neurasthenoid. Paresis may be confused with hypochondria and melancholia. Same factors distinguish it from hypochondria as from neurasthenia. Physical symptoms to be early looked for: Visual field may show some contraction at a very early period, but there is no retinal hyperesthesia, as in neurasthenia. Sequence of physical signs immaterial. The "paretic manner" so hard to describe, precedes all other physical signs, consisting of slight changes in facial expression and gesticulation: "that something in the eye, the face, the gestures, the words, which reveal that the patient is not in close and accurate touch with his surroundings, with his business affairs, with the fact of his illness, or for that matter, with any subject." Significance of specific history obvious. Very difficult to differentiate from alcoholism, for alcoholic excess is one of the

early symptoms of paresis. In alcoholism obtusion to proprieties and moralities soon cease after discontinuance of the alcohol. If alcoholic dementia exists, it is of the confusional type. General appearance of alcoholism indicative. In brain syphilis symptoms of organic lesion, diffuse, or limited, without quantitative mental changes. Early differentiation between tabes and paresis may be impossible, for many cases beginning apparently as tabes end in paresis. Absence of mental symptoms distinctive. Careful observation will generally lead to diagnosis in neurasthenoid stage.

3. *Comparative Frequency of General Paresis.*—Paresis, a disease known only about eighty years, being first recognized in this country at McLean Asylum by Dr. Bell in 1843. Statistics collected from various sources since 1849 show there has been a gradual increase from 1.5 to 12 per cent. at present of all admissions, and is everywhere from 5 to 8 times more frequent in men than in women, varying according to the urban or suburban character of the patients in the institution. The London Asylum reports show a percentage of 12 and 6 men to one woman affected, as well as that more married than unmarried people have the disease. It affects all classes and representatives of all vocations are found among its victims. Data from the Manhattan State Hospital East show a total average duration of a little more than two years and that native Americans are more prone to the disease than foreigners. It is further shown that the disease is rare before twenty and after sixty. From all sources of information it is found that this disease affects about 8.75 per cent. of all insane, is most frequent between thirty and fifty, is gradually increasing in frequency, men seven times more liable to the disease than women, is invariably fatal, and lasts less than two and a half years. Is nearly twice as frequent in a city population as in that of the rural districts. No special class subject to the disease, but wherever mental stress is combined with more or less hereditary influence. Overwork, sexual excesses, alcoholism, irregular mode of life, sunstroke, and cerebral traumatism seem to be special factors in its production.

4. *Treatment of Paresis; Its Limitations and Expectations.*—Neurological, histological and pathological findings present a barren field of limitations with respect to therapeutics. Pathological research with the microscope has nearly reached its limit, while physiological chemistry may be able to solve many clinical problems as yet beyond our ken. Metabolic changes must precede visible structural changes is a self-evident fact, and these metabolic changes are chemical within the molecules. From the etiological standpoint we are again limited therapeutically, as antisyphilitic treatment has generally proven futile. Heredity is of minor importance as an etiological factor. Alcohol may be a contributing cause by lowering the normal resistance to morbid influences, and therefore etiology affords no guide to treatment. The expectations of treatment offer an interesting field. While the relation between syphilis and paresis is unknown, it is probable there is some intervening factor or condition uniting them, for without syphilis, mental stress, excitement and excesses lead to neurasthenia, while the syphilitic neurasthenic generally becomes a paretic. A state of toxemia seems in all probability to represent this intervening condition. Drs. Mott, Kraepelin and Wernicke recognize the toxic agency in the etiology of paresis, which has induced a degeneration of the neurone, and this toxic agency may be of bacterial origin. It is a well established fact that auto-intoxications disturb the intimate harmony of brain cell metabolism, but this field is still one largely unexplored. Dr. Bruce and Dr. Ford Robertson have recently called attention to the exacerbations of this disease being accompanied by gastric and intestinal disorders, rise of temperature and hyperleucocytosis. By injecting serum obtained from a paretic during a remission Dr. Bruce claims to have effected a complete remission in two cases. Dr.

Bruce's conclusions are: "(1) General paresis is a disease directly due to poisoning by the toxins of bacteria, whose point of attack is through the gastric and intestinal mucous membrane. (2) The poisoning is probably a mixed poisoning, but the bacillus coli is apparently one of the noxious organisms. (3) The result of treatment with a serum taken from a case of general paresis in a condition of remission and injected subcutaneously into an early progressive case, points strongly to the fact that some form of serum treatment is the proper one." Dr. Robertson believes this disease depends on the occurrence of chronic toxemia of gastro-intestinal origin, due to a failure of the natural alimentary bacteria, thus giving rise to toxins whose absorption leads to vascular changes in the brain and so degeneration of the nerve elements; syphilis merely altering the natural immunity, and it is along this line Dr. Cowles believes that future researches may lead to alleviation of this disease.

5. *Heredity*—With a Study of the Statistics of the New York State Hospitals.—After defining the various forms of heredity and giving the statistics bearing on heredity from the several State Hospitals of New York, he summarizes by showing heredity in 25.8 to 39.7 per cent. of all cases. Nothing new is offered as to nervous diseases and the article concludes with a classification of nervous disease under direct, indirect heredity and those with no heredity.

6. *Senility and Senile Dementia.*—Senile cases constitute about 23 per cent. of the admissions to the Willard State Hospital. The writer recites the signs of senile decay, both mental and physical, in that there is a general blunting of the mental functions and a deterioration of the physical processes, both of which may remain within normal limits. There are wide variations as to the age when these changes appear and in the rapidity of their progression, yet sixty is regarded about the average. Temperament and diathesis are the prime factors which may cause senility to become of a pathological nature, as they largely govern the individual's resistance to pernicious factors, like the infections and toxins, and they thus induce senile dementia. A careful examination will generally be competent to separate normal senility from senile dementia, and of its symptoms the most important, aside from hallucinations and fixed delusions, are slight exaltation and restlessness, degraded, filthy habits, erotic tendencies, destructiveness and marked perversity in conduct. Insomnia may also be a prominent symptom.

7. *The Elimination of Indican, Acetone and Diacetic Acid in Various Psychoses.*—Indican seems to be increased in depressive states, particularly when attended by auto-intoxication, and its elimination decreases when the latter condition is overcome. The author gives a ready method for determining the relative amount of indican. The deductions of different writers are summarized as to the significance of indican, acetone and diacetic acid and their excess in the urine is generally accepted to be an indication of defective metabolism and coincident with auto-intoxication. Excess of acetone is said to occur in states of fear, but this the author has been unable to confirm. The abstracts of 26 cases are given, all of which have depressive phases, and a summary shows that indican is found to be coincident with katatonic and epileptic stupor, akinetic forms of dementia præcox and general paresis, as well as in alcoholic depression and depressive phases of the manic-depressive psychosis, or any disease where akinesis is a prominent symptom. On the other hand there is a lessened elimination of indican in katatonic excitement, exhilaration of general paresis, as well as in exalted states of dementia præcox and in the manic phases of the manic-depressive psychosis, or in other words in states of hyperkinesis. These variations in indican elimination are proven to be wholly independent of stools, diet and weight. Acetone was found in all the cases except epileptic

stupor, diacetic acid in melancholia, the akinetic conditions of dementia præcox and general paresis, alcoholic hallucination with fear. There appears to be no definite relation between the production of these bodies and the psychoses.

8. *Studies in the Manic-Depressive Insanity.*—Careful and systematic study of these cases reveals the presence of certain symptoms which form the basis of a common clinical complex whose variations are much more quantitative than qualitative changes. Use of the terms "mania" and "melancholia" smacks of casuistry, and the careful study of these conditions discloses their intimate relationship, the manic states generally being preceded by depression. Several cases are cited to elucidate this fact and they show that manic and depressive conditions are not antithetical. Further special study should be made of such cases as to blood pressure and the changes in the urine during the periods of excitement and depression.

McCorn (New York).

### REVUE NEUROLOGIQUE.

(Vol. X, 1902, No. 11, June 15.)

1. Primary Bronchial Cancer, followed by Miliary Carcinosis with Polyneural Complications. J. Oberthur.

1. *Polyneural Complications in Bronchial Cancer.*—An account of what the author believed to be a case of primary carcinoma of the left lung, having a bronchial origin, which rapidly invaded the right lung. The epithelial elements entering the general circulation through the pulmonary vein, were thus distributed throughout the entire system, and the cancer of the lung was most highly favored by a corresponding infection of the entire organism. The rarity of such a development, the large number and great variety of the metastases, the absence of any large ganglion accessible to palpation, the preponderance of motor and sensory phenomena, the difficulty of examination, the unusual progress of the disease, all these factors easily explain why a satisfactory diagnosis was impossible during life. The lesions of the neuro-muscular system were of two kinds; beside the ordinary lesions of cancerous cachexia, lesions of a mechanical nature were also present, on the one hand considerable compression and destruction of the motor and sensory nerve endings, and on the other hand general cancerous muscular inflammation, which together gave a particularly painful character to the polyneural complications of this peculiar pathogenesis.

Jelliffe.

### BRAIN.

(Vol. 25, 1902, No. 98, Summer.)

1. On Pachymeningitis Hæmorrhagica Interna. J. O. W. Barratt.
2. Case of Tubercular Tumor of the Spinal Cord in a Child Two Years Old. W. K. Hunter.
3. A Contribution to the Pathology of Acute Insanity. David Orr.
4. Uniradicular Palsies of the Brachial Plexus. E. Farquhar Buzzard.
5. A Case of Sclerotic Atrophy of Cerebrum and Cerebellum, Familial Type, Occurring in a Boy. J. Mitchell Clarke.
6. The Pathology of So-Called Acute Myelitis. H. Douglas Singer.
7. A Case of Acromegaly under Observation for Five Years, with Charts of the Fields of Vision. F. Richardson Cross.

1. *Internal Hemorrhagic Pachymeningitis.*—The author summarizes his observations as follows: (1) Intravascular separation of fibrin is an apparently constant event in subdural membrane formation. (2) Subdural membranes are free from micro-organisms as revealed by staining methods. The results of attempted culture upon agar, though not always negative, indicate that during life such membranes are free from bacteria. (3) Experimental implantation of false membrane into the subdural space of cats fails

to set up a progressive process of membrane formation. The few changes which occur outside the operation area are comparable to a process of repair. Finally the view may be advanced that pachymeningitis hæmorrhagica interna is a morbid process, in which the essential lesion is a separation of fibrin, usually within the blood vessels, upon which the other changes in this condition are largely if not chiefly dependent.

2. *Tubercular Tumor of the Cord.*—This is an interesting contribution particularly to the study of fiber degenerations with too many details to permit abstracting.

3. *Pathology of Acute Insanity.*—Dr. Orr presents the results of the examination of the nervous system of six patients with acute insanity dying of exhaustion. The appearances found in the cerebral cortex, fronto-motor area, and in the cord (Case 1) were slight changes affecting the edge of the chromophile bodies generally throughout the cell, and others showing atypical central chromatolysis. In the cortex the nucleus of the small pyramidal cells stains very deeply and diffusely. There are few cells in the gray matter of the cord which color deep blue and appear homogeneous. In the posterior root ganglia cells were profoundly altered by chromolytic changes. Two chief types of alterations are described as very characteristic. The first type is described as the large type in which the chromophilic elements are small and granuliform, both around the nucleus and in the central parts of the cell generally. Towards the edge the chromophile granules may either be found of a slightly larger size or there may be a distinct single row of large, slender elongated chromophile elements extending completely round the periphery of the cell. Elsewhere the granules are exceedingly small and densely crowded. The nucleus is central and is surrounded by a clear zone—the perinuclear space. The second type, the medium sized, with larger granules. The chromatolysis in both cells is varied. In the first the small granules break up into still smaller ones, lose their affinity for the stain and disappear and achromatosis ensues. Nucleus usually central but may be eccentric. In the second type the chromatolysis may be central, the granules break down to minute dust-like granules. The nucleus becomes eccentric to the least degenerated side. Vacuolation also occurs. In cases 2 and 3 similar cell changes were described. In case 4 a dissolution of the chromophilic material in the cortical nerve cells had taken place and in the body of the cell a faintly stained network can be perceived. The nucleolus is vacuolated. In case 5 the cortical changes as in 4. Case 6 showed similar changes to case 1. Degenerated fibers in the cord were constant.

4. *Uniradicular Palsies of the Brachial Plexus.*—E. F. Buzzard discusses, (1) the histories of six cases in which the symptoms pointed to a lesion of one of the spinal nerves forming the brachial plexus; (2) he discusses the sensoryand motor phenomena produced by such a condition; and (3) he considers the anatomical site and pathological basis of the morbid process concerned. From his study he concludes:

(1) There exists a clinical group of cases in which the symptoms strongly suggest a more or less complete destruction of functional continuity in one of the spinal nerves forming the brachial plexus: (2) the objective disturbances of sensibility resulting from this lesion closely conform to those observed by Sherrington after cutting a posterior root in a monkey. They differ in some respects from the anesthesia produced by the division of a peripheral nerve; (3) the anesthesia is largely disassociative in character, and does not occupy the whole cutaneous area to which the afferent fibers of the spinal nerve are distributed; (4) the objective hypesthesia is not associated with any subjective sensation of numbness, and is consequently overlooked by the patient; (5) the atrophy of the affected muscles is in excess of what might be expected in view of the fact that many of the

muscles receive fibers from two or more spinal nerves; (6) the condition generally occurs in persons who have at some time been subjects of a disease affecting the cardio-vascular system. The morbid process is probably vascular in character, and may be sudden or gradual in its onset.

5. *Sclerotic Atrophy of Cerebrum and Cerebellum of Familial Type.*—J. Mitchell Clarke describes a case of a boy of seven and one-half years, who clinically may be grouped as intermediate between a case of cerebral diplegia of post-natal origin, and a hereditary cerebellar ataxia with the occurrence of a peculiar form of blindness due to a special incidence of the disease upon the occipital lobes. The case seems unique, but is too detailed to admit of full abstracting.

6. *Pathology of So-Called Acute Myelitis.*—H. Douglas Singer gives a brief historical résumé of the pathology, describes the morbid anatomy in two cases of acute course, and presents some remarks on the etiological and clinical features of the disease. He maintains that:

(1) So-called acute myelitis is found on microscopical examination, in the majority of recorded cases, to be not inflammatory, but due to thrombosis of spinal vessels. (2) That by far the most common cause of this thrombosis is syphilitic arteritis; and that senile arterial degeneration forms a considerable proportion of the remaining cases; (3) this view as to the pathology is confirmed clinically by the analogy between this disease and cerebral thrombosis. In conclusion he urges that the recognition of thrombosis of spinal vessels as the most common cause of the symptom-complex described as acute myelitis is not merely of academic interest, but may be of practical value. It is possible that prompt treatment, in many cases presenting premonitory symptoms and giving a history of recent syphilis, may ward off an attack of this nature. For this reason alone, to say nothing of the confusion which has arisen from the indiscriminate use of the term "myelitis," he advises that the nomenclature used by Dr. Bastian should be adopted, and that these cases be classed as "thrombotic softening of the spinal cord," or, more simply, "spinal thrombosis"—a name which at once suggests the analogy with cerebral thrombosis.

7. *Acromegaly.*—F. Richardson Cross describes a patient who has been under observation five years and gives complete charts of the visual fields that showed that in 1895 no definite serious impairment of sight was present. Acromegaly was not then pronounced. In 1897, with complete establishment of the disease, there was blindness in the temporal field of the right eye and almost complete loss of sight in the left eye. The degrees of the blind portions in the visual fields of both eyes have progressively lessened with the improvement in the general health. JELLIFFE.

### ARCHIVES DE NEUROLOGIE.

(Vol. 14, 1902, No. 81, September.)

1. General Paresis from Data Compiled at the Psychiatrical Clinic of the University of Moscow: by S. SOUKHANOFF and P. GANNOUCHKINE.
2. Corneal Depression and Serious Encephalopathic States. B. PAILHAS.
3. A Case of Hysterical Breast. VIALLON and ALOMBERT.
4. Hysteria of Saint Theresa.

1. *General Paresis.*—This article is a statistical study of 682 cases of general paresis which have been treated in the Moscow Clinic between the years 1887 and January 1, 1901. The total attendance during this period was 3,916. Of the paretics 590 were men and 92 were women. This shows that a fifth of all the cases were paretics; the relation of the male paretics to the total number of male cases was 1 to 4; while the female paretics were only 6.5 in 100. Their tables show that the number of paretics has increased in the last few years, not only absolutely, but what is of more significance, relatively as well. The number of female paretics in-

creased in the past four years from 5.54 to 8.09 per cent. They consider that mental disease is twice as frequent in men as in women, and that this ratio has not been materially altered in more recent years; bearing this relation in mind their data show that the relative number of female paretics has increased very much more in the past four years than the number of male paretics.

The largest number of cases of paresis occur between the ages of 31 and 40; most of the male paretics occurring during the second half of the period and most of the female in the first half. Regarding the influence of occupation they conclude that farming and country life do not predispose to general paresis. They found that in 75 per cent. of the cases there was present a psychopathic or neuropathic hereditary factor, and in almost one-half of all the cases there was a history of alcoholism on the part of father or mother. These figures correspond to those given to show the influence of heredity in all mental disease, and hence simply designate general paresis as being in this respect in harmony with psychoses in general. Syphilis was positively present in 61.54 per cent. of the males and 20.9 per cent. of the females; very probable in 9.23 per cent. of males and 25.37 per cent. of females; possible in 9.61 per cent. of males and 14.92 per cent. of females; denied in 19.61 per cent. of males and 38.81 per cent. of females. The time elapsing between the syphilitic infection and the development of general paresis in 90 per cent. of the men varies from six to twenty years. The longest period was thirty-three years, the shortest, three years. Alcoholism in one degree or another played a rôle in 80 per cent. of the male cases and 40 per cent. of the female.

Among 497 paretics they observed forms of dementia in nearly 50 per cent; the form of mania in 34 per cent; melancholia in 6 per cent. and other forms such as paranoia, circular, *etc.*, much more rarely. The patellar reflex was found to be exaggerated in one-half of the cases, and absent in one-fifth of the number (of males) and one-sixth (of females).

The pupils were unequal in 73 per cent. of males and 66 per cent. of females; they were equal in 26 per cent. of male paretics. and in 36.7 per cent. of female paretics. The pupils did not react in 82 per cent. of the male cases and in 77 per cent. of females. There was reaction in 17 per cent. of males and in 22.5 per cent. of females. Apoplectiform attacks occurred much more frequently than epileptiform.

2. *Corneal Depressions.*—Pailhas calls attention to the depressions in the cornea described by Brière de Boismont as occurring in the eyes of insane during an attack of acute delirium and notes that they are present in patients suffering from serious encephalic conditions. They are not caused by any appreciable anatomical lesions. The corneal depression is unstable, changes its location, appears and disappears. Although in one case it developed within an hour after a violent cranial injury, as a rule it appears only at time of marked psychical depression, of acute delirium or of encephalitis. Pailhas thinks that lesions of the cerebral nerve centers exercise in some unknown way an indisputable morbid influence on the cornea, and that the prognosis in cases of encephalitis, acute mania or cranial traumatism in which corneal depression develops, is unfavorable either as regards life or as regards dementia following depressed psychopathic states.

3. *Hysterical Breast.*—Viallon and Alombert report the case of a young girl, eighteen years old, presenting some of the stigmata of hysteria, who at time of menstruation has very marked engorgement of the breasts. At the same time she is in a very nervous and excited state. The swelling persists for a few days during which it is alternately increased and diminished in size, but is at its maximum during the menstruation or for a few days preceding it. The breasts are painful and tender when swollen. No induration. Nipples retracted. No fever. The mental state on one occa-

sion was almost maniacal. This condition of the breast has been ascribed by some writers to the influence of irregular and painful menstruation. The menstruation of the patient was of such unhealthy character. The enlargement differs from the normal physiological swelling of the breasts at time of menstruation simply in its much greater size and its being accompanied with pronounced and well developed hysterical manifestations.

4. *Hysteria of Saint Theresa.*—Continuation of article begun in preceding number. STRAUSS (New York).

### ARCHIVES D'ELECTRICITE MEDICALE.

(1902, No. 15, July.)

1. The Electrolytic Treatment of Xanthelasma. P. PAUSIER.
2. A Study of Galvano-Faradisation. CLUZET.
3. How Should the Electric Treatment be Applied in Basedow's Disease, and What Results can be Expected? M. MALLY.
4. The Electric Treatment of Infantile Paralysis; A Clinical Study. E. ALBERT-WEIL.
5. The Laws that Determine the Opacity of Different Antiseptic Powders to X-rays. DARCOURT.

1. *Treatment of Xanthelasma.*—A description of two cases of xanthelasma which were successfully treated by the author, who employed for the active negative electrode several needles introduced into the diseased areas of skin. After five or six applications of a current of from 6 to 10 M. A. the patches completely disappeared.

2. *Galvano-Faradisation.*—A lengthy, detailed paper in which galvanofaradisation is studied in its physical, physiological, and therapeutical aspects. The author concludes that galvano-faradisation, thanks to the combination of the motor, electrotonic, and catalytic actions of the composite currents, presents real advantages over galvanism or faradism used individually.

3. *Electrical Treatment of Basedow's Disease.*—After discussing the various clinical forms of exophthalmic goiter and the various symptoms calling for the application of special currents, the author summarizes as follows: "(1) Electricity in all its forms can be of use in the treatment of Basedow's disease. (2) We have endeavored to show that a rational electric treatment is conceivable which is adaptable to every form of Basedow's disease. It consists, in fact, of constantly taking into consideration the general and symptomatic state of the patient; a univocal treatment of Basedow's disease would be nonsensical therapeutics. (3) Static electricity can be used to advantage on patients with sluggish nutrition. In the opposite condition this agent is not tolerated, and its employment may cause harm. (4) Faradism may be used to combat individually all the morbid symptoms of Basedow's disease: dilatation of the carotids, thyroid hypertrophy, exophthalmos, muscular paresis, and tachycardia. Its judicious employment always produces a diminution, at least momentary, of the distressing symptoms; intolerance is never noticed. (5) Galvanism is more restricted in its application; this form of electricity, whose efficacy is not doubtful, can be used in certain cases, alone or in combination with faradism.

4. *Treatment of Infantile Paralysis.*—In this article the author describes his method of treatment of anterior poliomyelitis and gives the results obtained in twenty cases, some very recent and others of long standing. The method recommended is to employ in the immediate post-febrile period a mild descending stabile current, using a broad electrode on the back and suitable sized vessels filled with water as electrodes for the paralyzed extremities. At the end of the third week a rhythmically interrupted galvanic current is to be used and as soon as the regenerating muscles respond to faradism, that form of current is to be used.

5. *Opacity of Antiseptic Powders to X-rays.*—The author summarizes his results as follows: (1) Compound substances that contain a simple substance of high atomic weight are opaque to X-rays. (2) The higher the atomic weight of the simple body, the greater the degree of the opacity. (3) For compounds that contain the same simple substance of high atomic weight, but in different proportions, the opacity is directly proportionate to the percentage of the simple substance present.

R. H. CUNNINGHAM.

(115, 1902, August.)

1. On the Present State of Electrodiagnosis. J. CLUZET.
2. A New Example of the Numerical Application of the General Laws Governing the Transparency of Matter to X-rays. L. BENOIST.
3. The Action of Continuous Currents on Sclerotic and Cicatricial Tissues. STÉPHANE LEDUC.
4. On Electro-Diagnosis upon the Bared Nerve in Man. J. BÉRGONIE.
5. An Historical Sketch of Electro-Therapy. E. ALBERT-WEIL.

1. *State of Electrodiagnosis.*—A long detailed paper unsuitable for abstraction. The author, however, considers that the method of exploration by means of condenser discharges yields the best results and is the method to employ in electrodiagnosis.

2. *Transparency of Matter to X-rays.*—A short paper correlating the results of Dr. Darcourt (see the July abstract) with those previously published by the author, from which, by means of a given formula, the opacity of various substances to X-rays can be readily calculated.

3. *Action of Continuous Currents.*—After relating in detail his results in six patients with cicatrices, giving rise to more or less deformity and disability, the author attributes the beneficial action to the fact that continuous currents in passing through the organism give rise to a double current of ions, the hydrogen, metals and metallic radicles descending with the current, and the hydroxyl, OH, and the acid radicles ascending with the current. The therapeutic action is undoubtedly due to the production of these movements.

4. *Excitation of Bared Nerves in Man.*—On certain occasions it becomes necessary to make an absolutely definite diagnosis and prognosis in disease of or injury to peripheral nerves, and owing to difficulty of doing so when the nerve is excited through the overlying skin and other tissues, the author recommends exposing the nerve and stimulating in the usual manner employed by the physiologist. He concludes:

"(1) It is easy to investigate the state of the irritability of a nerve exposed during operative intervention. (2) This research gives the most valuable indications regarding the diagnosis and the prognosis. (3) Inexcitability determined in this way points to a very grave prognosis."

R. H. CUNNINGHAM.

(1902, 116, September.)

1. On Accidents due to X-rays. ONDIN (de Paris).
2. International Congress of Electrology and Radiology held at Bern, September 1-6, 1902.

1. *X-Ray Accidents.*—In this extensive paper occupying thirty-three pages, Dr. Ondin presents a very thorough account of the pathology and treatment of radio-dermatitis in its acute and chronic forms. As regards the treatment practically nothing has been found so far that prevents the development of the burn after prolonged exposure. The author considers that the X-rays alone produce the dermatitis and that metallic screens merely protect in accordance with their transparency to X-rays. To protect his own hands the author uses fencers' gloves, containing a padding of fine brass wire.

R. H. CUNNINGHAM.

(1902, 117, October.)

1. The Influence of the Galvanic Current upon Muscular Force. SCHNYDER.
2. The Production of Sleep and of Local and General Anesthesia by Intermittent Low-Tension Currents. STÉPHANE LEDUC.
3. A New Form of the Electrolytic Interrupter.
4. Radioscopy and Radiography of the Abdominal Organs. A BÉCLÈRE.
5. On Surgical Electrolysis and Galvano-Caustics. TH. GUILLOZ.

1. *Galvanism and Muscular Force.*—From his results the author concludes that "Voltaic electrisation of the vertebral column exerts a favorable influence upon muscular force, which is manifested less by the increase in work expressed in kilogrammeters than by the modifications produced in the form of the fatigue curves. By the greater number of ordinates and by their more gradual descent, the S-like form of the curve very strongly points to the preceding fact. But this favorable influence upon muscular force is produced not by electric stimulation only. Another stimulus, acting under similar conditions, such as a sinapism, has nearly the same influence upon muscular force."

2. *Production of Sleep, etc.*—An experimental research upon the lower animals in which the negative electrode is applied to the head and the broad positive to the back. A very rapidly interrupted continuous current of an E. M. F. of 16-30 volts is applied for a short time, the voltage being rapidly reduced. The author finds that it is possible to produce by interrupted low tension currents painless, instantaneous and complete inhibition of the cerebral hemispheres in animals, while the respiratory and circulatory centers are unaffected. Thus one produces a quiet and regular sleep that can be prolonged for several hours, and a general and complete anesthesia. Sleep occurs or disappears rapidly accordingly as the circuit is closed or opened. No after-effect follows the sleep. Prolonged and repeated sleep produces no apparent change in health.

3. *Electrolytic Interrupter.*—An illustrated description of a modified Wehuett interrupter for use with alternating currents.

4. *Radioscopy of Abdominal Organs.*—A lengthy detailed paper devoted to the radioscopy and radiography of all the various organs of the abdomen and thorax. The author concludes that the X-ray method of examination constitutes an exceptional means for the investigation of the nervous system, a very valuable method for the abdominal organs which is particularly applicable for finding urinary calculi, and is a very admirable method for the physical examination of the thoracic organs. It is destined to rank in current practical medicine with auscultation and percussion, and to occupy in time a more important place.

5. *On Electrolysis.*—A lengthy paper unsuitable for abstraction, that discusses electrolysis in its physical aspects and in its therapeutical applications.

R. H. CUNNINGHAM.

## MISCELLANY.

REPORT OF A CASE OF PROGRESSIVE MUSCULAR ATROPHY (SPINAL) WITH MENTAL SYMPTOMS. R. Edward Garrett (Maryland Med. Jour., Oct., 1902).

Subject of report female, fifty-three years old. Patient for seven months at Maryland Hospital for the Insane. Symptoms at date of admission not sufficiently marked to justify positive diagnosis of progressive muscular atrophy. Has chronic nephritis. No hereditary predisposition. No luetic nor rheumatic history. Slight transitory pains about chest and shoulders. Case one of "upper arm type" with pronounced atrophy of muscles of trunk, particularly of arms, shoulders and chest. No changes observed in the legs. Feet and ankles edematous. Protrusion of head forward. Characteristic kyphotic attitude. No paralysis, but considerable weakness of affected muscles. No disturbance of speech, nor loss of control of sphincters. Gait normal. Patellar reflex absent. Pupils equal, reacting to light and accommodation. No optic nerve changes. No bulbar symptoms. At times choreiform movements of upper and lower extremities. Fibrillary tremor not detected. Mental symptoms considered to be part of the general disease, due to extension of spinal lesion to the brain. Patient feeble-minded, forgetful, unable to concentrate the attention. Vocabulary reduced to a few words. Mental impressions retarded.

J. E. CLARK (New York).

THE BLOOD, FROM A CLINICAL ASPECT. J. G. Emanuel (The Birmingham Med. Rev., June, 1901).

In tuberculous meningitis there is generally a leucocytosis. Given a doubtful case of typhoid or tuberculous meningitis, a raised count would negative uncomplicated typhoid and would be in favor of tuberculous meningitis. J. E. CLARK (New York).

A CASE OF PRIMARY PNEUMOCOCCUS MENINGITIS SIMULATING PUERPERAL ECLAMPSIA. Thomas Wilson and J. G. Emanuel (Birmingham Med. Rev., April, 1902).

The authors report an interesting case of pneumococcus meningitis, with autopsy, which had been previously diagnosed puerperal eclampsia. The patient, multipara, six to seven months pregnant, admitted to the Birmingham General Hospital in a comatose condition. For several days had complained of headache, followed by convulsive seizures and unconsciousness. Albumin and sugar present. No edema. Heart and lungs negative. Death on fifth day after onset. Post-mortem examination disclosed a purulent meningitis due to pneumococcus infection. Brain 41 ounces by weight. Surfaces of cerebrum, cerebellum, pons and medulla covered with purulent exudate. No changes observed in gray or white tissue upon gross section. Ventricles normal as to distention. No signs of tubercle, middle ear disease nor pneumonia. No septic focus. Bacteriological examination of exudate revealed diplococci resembling Fraenkel's pneumococci. Glycosuria was attributed to the meningitis in association with an enlarged and fatty liver. J. E. CLARK (New York).

MENTAL DISTURBANCE DURING THE PUERPERIUM. W. H. Hattie (Montreal Med. Jour., Sept., 1902).

Ten per cent. of females admitted during past three years at Nova Scotia Hospital for treatment of "so-called" puerperal insanity. The writer, while allowing due consideration for inherited predisposition, believes that a large proportion of cases result from causes (toxic or infectious) which are generally preventable at this period. Prophylaxis in respect to the maintenance of healthy bodily nutrition, avoidance, so far as possible, of nervous stress, antisepsis, *etc.*, are recommended.

J. E. CLARK (New York).

THE BLOOD IN CERTAIN CUTANEOUS, NERVOUS AND MISCELLANEOUS DISEASES, WITH REMARKS UPON THE ORIGIN AND SIGNIFICANCE OF THE EOSINOPHILES. Thomas R. Brown (Maryland Med. Jour., July, 1902).

In twelve observations, upon two cases of chorea, the writer states the total number of leucocytes was normal in every case, while the eosinophiles varied between 5.2 and 9.5 per cent., that is, there was always a distinct, and on some occasions a definite increase in these cells. In connection with Neusser's views, that "the supply of eosinophile cells in the blood is controlled by the sympathetic nervous system, and eosinophilia is the expression of sympathetic nervous irritation," the above is of especial interest. In three observations, on three cases of angio-neurotic edema, both leucocyte count, and percentage of eosinophiles, were practically normal. While in two of the three observed cases of epilepsy, also reported upon, there was a leucocytosis, one slight, the other quite marked, 18,000. The eosinophiles were present to an extent of one per cent. in two of the cases, and five per cent. in the third, a case with a leucocytosis of 10,500 and associated with incipient senile dementia. Burr and Murphy, separately, in chorea, have shown that an anemia is usually present. Murphy believes that chorea is the cause, the blood changes being the effect.

Krainsky considers the blood in epilepsy to be increased as to its toxicity. Herter holds divergent views. Jenks believes there is a marked increase of large mononuclears, prior to the epileptiform seizures, while Capps and Burrows have found that a leucocytosis, often of a very marked degree, is associated with convulsions, whatever the cause.

Kappert, as a rule, in organic and functional nervous disease, found a moderate eosinophilia. Neusser describes a similar condition in various nervous disorders J. E. Clark (New York).

Post-Operative Insanity. Alex. Pilcz (Wien. klin. Woch., Sept. 4, 1902).

Of the small number of cases of insanity following surgical operation, some may be referred to a previous syphilitic infection, others to alcoholic poisoning, or suddenly enforced abstinence from alcohol. Chloroform or iodoform are possible causal agents. Senile patients sometimes develop mental disturbances as a result of loss of blood, narcosis, antiseptics and other irritations. Among children, except in cases of sepsis, insanity is practically unknown. Among cases with definite degenerate disposition or bad heredity, insanity not infrequently develops. Hysteria is especially liable to give trouble. The general condition at the time of operation is the most important question in the production of acute post-operative insanity. Cachexia of any sort, malnutrition, gastro-intestinal disorders have definite influence. Psychical factors are important, such as fear of operation, excitement, or mental depression.

After certain operations, such as castration, the loss of an "internal secretion" has been held to be the cause of mental disturbance. The author believes that the cause is more probably reflex psychoses, such as might have occurred from other circumstances. In most cases iodoform poisoning or slight or severe sepsis is the cause of mental disturbance. In a fatal case of Voisin, after ovariotomy with wild delirium, an acute hemorrhagic meningo-encephalitis was found at autopsy, though the wound was in good condition. A case of Seeligman, of acute delirium following a gynecological operation, large doses of opium had caused constipation and secondary auto-intoxication. Auto-intoxication and uremia are active and not infrequent causes. Excessive pain sometimes causes transitory psychosis. The clinical type of the insanity may vary from mania to paranoia. Iodoform poisoning simulates acute delirium. The hysterical cases develop hysterical delirium, "folie des dégénerés." Old people develop typical senile insanity. W. B. Noyes (New York).

# Book Reviews

PSYCHIATRIE FÜR AERZTE UND STUDIRENDE. Bearbeitet von Dr. med. Th. ZIEHEN. O. Professor an der Universität Utrecht. Zweite Auflagle. S. Hirzel. Leipzig. G. E. Stechert, New York. Sixteen marks.

In the application of good common sense physiological psychology to the study of psychiatry this work stands out as one the ablest exponents. Founded as it is largely on the author's well known and widely employed "Leitfaden" the two make a coherent whole as far removed from the old-fashioned metaphysical psychiatry as it is possible to remove any study that involves so much of the terminology of that pursuit of the earlier students of philosophy. On the point of classification we believe the author to be extremely logical, particularly from the standpoint of the physio-psychological school.

The work is divided as most of our works on psychiatry are into the General and Special portions. Under the head of General Symptomatology Ziehen discusses in order, disturbances of sensation, perception, memory, intellectual tone, and feeling tone, association of ideas and disturbances of conduct. Bound intimately with these general sub-chapters the somatic accompanying symptoms are taken up.

Chapters in the General Development of the Psychoses, Diagnosis, Etiology, Prognosis, Pathological Investigations and of Therapeutics close the first portion of the volume.

Under Special Psychopathology there are considered (1) the Psychoses without Defect of Intelligence, including the simple affective psychoses, mania and melancholia, the intellectual psychoses with various types of paranoia, confusional states, fever and toxic deliria, and the psychopathic constitution; (2) the Compound Psychoses, periodic and nonperiodic, the defectives, born and acquired. Following these are a few pages devoted to general considerations.

It is impossible to enter completely into an analysis of the author's various lines of demarcation. They have their analogues in the cases seen in private and hospital practice, and the systems outlined are remarkable in the acuity of their differentiation.

The work is deserving of a carefuly study, and it can be most heartily commended as an earnest and comprehensive work on psychiatry.

JELLIFFE.

PSYCHOPATHOLOGICAL RESEARCHES. STUDIES IN MENTAL DISSOCIATION. By BORIS SIDIS, M.A., PH. D., Director of the Psychopathological Laboratory. Published under the Auspices of the Trustees of the Psychopathic Hospital, Department of the New York Infirmary for Women and Children. G. E. Stechert. New York, London, Leipzig and Paris.

THIS is an extremely interesting and suggestive volume that merits an extended notice. The space at our command, however, does not permit us to do it full justice. There are eight separate studies in the volume as follows: Some General Remarks Concerning Psychopathological Research, by Boris Sidis; Mental Dissociation in Functional Psychosis, by Boris Sidis and William A. White, M.D.; Mental Dissociation in Alcoholic Amnesia, and "in Psychic Epilepsy," by William A. White; Mental Dissociation in Depressive Delusional States, by Boris Sidis and George M. Parker, M.D.; Mental Dissociation in Functional Motor Disturbances, by George M. Parker, and Mental Dissociation in Psycho-motor Epilepsy, by George M. Parker.

Sidis makes an interesting gradation of affections of the psyche into three great classes, the which pass into one another by imperceptible degrees, these he styles: functional psychosis, functional neuropathy and necrotic neuropathy. Disaggregation and disintegration, however, may be present in different degrees in varying groups, and neurone systems. His complete formula being expressed as follows: "The symptomatic side of disease, the total psychomotor aspect of the pathological process, is a function of location, number, and degree of dissociation or degeneration. The total complex of psychomotor manifestations depends on the location and number of neurone aggregates involved and on the stage or degree of the pathological process of disaggregation, dissociation or degeneration."

With reference to the attitude towards the insanities, he says: "Functional psychosis, functional insanities, should become a special research field of the psychopathologist. Functional psychosis is specially characterized by psycho-physiological disaggregation where synthesis is still possible. The only way of restoring the disturbed equilibrium is to bring about a synthesis of the disaggregated groups with the functioning systems of the upper active personality." To bring about these syntheses the author makes use of his intermediary states.

Throughout the book the recognition of the so-called subconscious life is imperative. Upon its existence the arguments are founded and the work in reality is an excellent study of the subconscious physio-psychological processes, well worth the reading. JELLIFFE.

ZUM STUDIUM DER MERKFÄHIGKEIT. Experimental-psychologische Untersuchung. Von DR. AUG. DIEHL, Nervenarzt in Lübeck. S. Karger, Berlin.

The study of the power of correct observation and firm memory grasp is of interest and of importance not only to the psychologist, but to the jurist, and the physician—not to mention the everyday business man.

From the psychiatric side there has been a distinct lack of criteria for the measurement of normal ability in this series of faculties. This lack the present study in part fulfills, and does so very well.

It is a psychological study primarily with a very practical trend, and is well worth permanent preservation in its present small monographic form. SMITH.

GENERAL PARESIS. PRACTICAL AND CLINICAL. BY ROBERT HOWLAND CHASE, A.M., M.D., Physician-in-Chief Friends Asylum for the Insane, *etc.* P. Blakiston's Sons & Co., Philadelphia.

The author's object in devoting an entire volume to this disease is distinctly sensible. The general practitioner does not recognize general paresis as soon as it can be recognized, and in consequence many calamities occur that might be averted. There can now be no excuse for the general practitioner with such an excellent manual as this for his guidance, since it has been from this practical point of view that the work has been written. The book is particularly rich in illustrative cases. These cover an immense field of experience, and put the reader *en rapport* with very dissimilar types of the affection.

It cannot fail to be of great service, even more than a much more technical presentation of the subject. S. E. J.

LES PARALYSIES DES NERFS PÉRIPHÉRIQUES, ET LA SYSTEMATISATION DE CES NERFS. Par DR. CHARLES VIANNAY, Ex-interne des Hopitaux de Lyon, Préparateur du Cours de Médicine Operatoire. J. B. Baillière et Fils. Paris, France. 1903.

This is a small monograph of 150 pages, bearing on the subject of peripheral paralysis.

The first half takes up a general study of the topography distribution and systemization of the chief nerves of the arm and leg distribution. This is discussed not from the purely anatomical standpoints, but from the side of its application in the study of peripheral palsies.

The second half deals with the clinical side of peripheral nerve affections, first discussing the generalities, etiology, *etc.* Radial, median, cubital and sciatic palsies are taken up in turn, the motor, sensory, and trophic disturbances each receiving special consideration.

The work is concise and practical and highly suggestive, and is well worth a careful reading. JELLIFFE.

THE FORCE OF MIND OR THE MENTAL FACTOR IN MEDICINE. By ALFRED T. SCHOFIELD, M.D., M.R.C.S. P. Blakiston's Son & Co., Philadelphia.

To those who have watched the trend in certain lines of philosophical development this volume will not come as a surprise. It is a logical outgrowth of much thought that has of late years been centered on things psychical.

Not many years ago, it was considered by some a sign of superior intelligence to deprecate this aspect of thought, but that time has gone by, and whereas the narrow-minded never really becomes other than one who has the "will to disbelieve" he now finds himself at a loss for his lack of understanding of now well recognized and true phenomena.

Coming to the volume at hand, however, is it one that will commend itself as an authoritative expression of modern day science on the subject of mind cure? In a sense it is not, but withal it is an extremely interesting, though heterogeneous, and, we fear, much jumbled, collection of opinions and facts, the which, be it said to the author's credit, is to him a palpable fact.

The book is divided into two parts. Part I. considers "The Action of the Mind in Causing Disease," and Part II. "The Action of the Mind in Curing Disease." In Part I. the author considers the force of mind, the unity of mind, psycho-pathology, mental factors in organic and other diseases, causes and symptoms of functional nerve disease, etiology of hysteria, and phenomena and illustrations of hysteria. The chapter headings of part II. are psycho-therapy, the *vis medicatrix naturae,* some varieties of mental therapeutics, illustrations of the curative effect of mind, mental therapeutics in functional nerve diseases, therapeutics of hysteria, and practical conclusions.

The work is largely made up of extracts and sentences from the published work available in the English language. There is almost no reference to and apparently little acquaintance with the vast literature of the French authors, who, of all the students of mental phenomena, have advanced the sum of human knowledge in this branch. Thus Charcot and Janet are not even mentioned in the author's extensive bibliography. Thus it may be seen that the work is more of a patchwork than an authoritative utterance, but it is an attempt in the right direction and is worthy of commendation. JELLIFFE.

# News and Notes

One of the three new buildings being erected at the University Hospital, at Ann Arbor, Michigan, is to be a psychopathic hospital. The earliest stages of all forms of acute psychoses are to be studied here. The State appropriated $50,000 for its erection and will complete its equipment with the best modern scientific apparatus. An annual appropriation will be made by the State for its future maintenance.

Dr. John Fitzgerald, Superintendent of the Rome Custodial Asylum, has resigned this position to accept the one of General Medical Superintendent of the four general hospitals of Kings County.

Dr. Veavianos, of Athens, Greece, has undertaken the editorship of a new journal of psychiatry and neurology and allied subjects.

The American Association for the Cure of Inebriety held its thirty second annual meeting in the hall of the Washington Home, Boston, Mass., on December 18, 1902.

A recent mental study of four hundred mendicants in Breslau showed the following: One-half of the entire number presented a neuropathic history, such as alcoholism (29 in 100), epilepsy, hysteria and various other psychoses. The intelligence was of a low order in all; (53 out of 100) had not obtained even a preliminary education; one-third showed arrested development, congenital or post natal. Acquired diseases were noted in 6 per cent.; general paresis was the most prominent of the psychoses. The majority (65 per cent.) were chronic alcoholics.

The managers of the New Jersey State Colony for Epileptics at Skillman report after making a State census that there is one epileptic to every five hundred of the State population, and therefore ask for a substantial increase in legislative appropriation to enlarge and improve the present institution, which cares for only eighty-five patients. Since 1898 nine patients have been discharged cured.

It is expected that the New York Commission of Lunacy will ask for a material increase of the annual maintenance fund for the State Hospitals under their care for the coming fiscal year. The appropriation for the purpose including the necessary building construction, was $4,165,886.66 for 1902, which was $340,000 less than for the year 1901. However, a large balance used from last year's funds is not available this year, hence the necessity of an increase.

Dr. S. Weir Mitchell is to deliver the University Day oration in commemoration of Washington's birthday at the University of Pennsylvania.

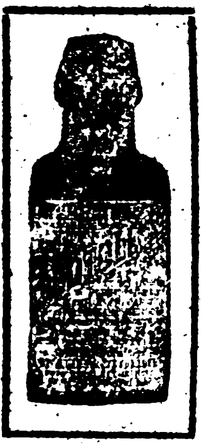
The committee selected by the last legislature of Pennsylvania, to erect the Homeopathic State Hospital, met in Philadelphia on December 10, to open bids and proposals for various sites for which the State appropriated $50,000. The coming legislative session is to be asked for a substantial sum to erect the buildings as speedily as possible to meet the long existent demand for such an institution.

The plans which provide for the administration building of the Maryland Asylum and Training School for Feeble-minded at New Owings' Mills have been completed and construction is to begin at once. The building is to be for administrative officers and teachers.

For State Hospital Co-operation.—The aims and plans of the Pathological Institute of the New York State Hospitals were discussed by Dr. Adolf Meyer in a paper which he read before the physicians on Ward's Island recently. The institute was found by Dr. Carlos Macdonald and reorganized last year. As to what must be accomplished Dr. Meyer said: "We must make the work of the physicians as medical as possible, abolish all regulations which derange their work; in short, all and everything that makes a young assistant put trivialities above medical accuracy. We must investigate how large a force is required to reach a standard of work on which we can agree. We must make somebody responsible for the accuracy and efficiency of the medical work which the superintendent cannot possibly supervise well enough where he is responsible for all the administrative burdens; and this person, or these persons, let us say the older members of the staff, must be officially obliged to be able to be helpers to the younger members, sources of instruction and stimulation." Dr. Meyer argues that medical schools do not provide the necessary training for actual hospital work and that assistant doctors in the hospitals come unprepared for special work. "Under these conditions," Dr. Meyer says, "the hospital system must help itself." It has been urged that the prime duty of the institute should be original research work and the discovery of better methods of prevention, treatment and cures. This, he says, is the ultimate object.

New Superintendent of Bellevue.—The Trustees of Bellevue and allied hospitals have invited Dr. William Mabon, the Medical Superintendent of the St. Lawrence State Hospital at Ogdensburg, to accept the position of Superintendent of the hospitals under their charge. Dr. Mabon graduated from the Bellevue Hospital Medical College in 1881. After several years spent in private and hospital practice, he entered the service of the State in 1887, having received the appointment of Assistant Physician at the Utica State Hospital for the Insane. He remained at Utica until October, 1895, when he was appointed Medical Superintendent of the Willard State Hospital. This hospital was in need of reorganization, and it was believed that Dr. Mabon's experience and training and executive ability qualified him for the work. In one year the hospital was placed upon a sound administrative basis, and in October, 1896, the managers of the St. Lawrence State Hospital at Ogdensburg called him to the Superintendency of that hospital to succeed Dr. Wise, who had been appointed President of the State Commission in Lunacy. Under Dr. Mabon's management, the reputation of the hospital as leading in all the best methods for the treatment of the insane has been fully maintained. With the consent of the State Commission in Lunacy and of the State and Municipal Civil Service Commissions, Dr. Mabon now returns to New York City, and will take charge of Bellevue and its allied hospitals on January 1, 1903. In view of his lay experience in the State service, and his proved administrative capacity, it is confidently expected that he will raise these hospitals to the highest state of efficiency. In addition to his executive duties, Dr. Mabon will also act as consulting physician to the Pavilion for the Insane of Bellevue Hospital.

Annex to School for Feeble-Minded Asked For.—The board of directors of the Pennsylvania Training School for Feeble-Minded Children has decided to petition the legislature for $150,000 to build an annex for housing unimprovable cases. It is proposed to place the annex five miles from the main building. Citizens of Delaware county do not favor the plan, as it means more property exempt from taxation. This county, it is stated, now has more land for public institutions exempt from taxation than any other county in the State.



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The Official Organ of

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The New York Neurological Society
The Philadelphia Neurological Society and
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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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Volume 30. February, 1903. No. 2

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## Original Articles.

# A CASE OF COLLOID DISEASE OF THE BLOOD VESSELS OF THE SPINAL CORD.[1]

By F. X. Dercum, M.D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASES, JEFFERSON MEDICAL COLLEGE, NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

The following case is unique in my experience and I have been unable to find any other like it in the literature. As the title indicates, it is one of focal lesion of the cord due to colloid disease of its blood vessels. The history is briefly as follows:

M. H., age forty-three, white; occupation, domestic.

*Family history*—Father and mother both killed by accident; otherwise negative.

*Previous history*—Has had the various diseases of childhood. Menstruation began at eighteen years, then ceased for a year, returned and occurred regularly every two weeks for two years. Subsequently it continued regularly every third week up to the present time. Suffered from displacement two years ago from which she recovered under treatment. Four years ago she had a slight attack of influenza, characterized by severe pains in neck, back and chest. About this time also was attacked by diarrhea which patient attributed to exposure to cold and wet. She characterized the diarrhea as uncontrollable. She continued to be up and around, however, and attended to her work. At the end of a year the diarrhea ceased and she began to slowly lose power in both hands, especially in the right. She was able, however, still to walk without difficulty.

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[1] Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

About a year later she began to have pains in the back, muscular rheumatism and stiffness of the joints. Her condition at the time of admission was as follows:

She stood with difficulty apparently because of stiffness in the joints of the legs and feet. There was stiffness of all of the joints, particularly of the smaller ones. There was a slight contraction of the middle and ring fingers of the right hand. Ankle-clonus was not noted, nor was there noted any sensory loss. The general physical examination was negative, save that both sounds of the heart appeared to be slightly roughened and there was a partial opacity of the left cornea, the result of scarlet fever.

Her condition presented from time to time little change. Examined a year later, it was found that the muscles of the shoulder had become distinctly atrophic, the fingers were contracted, the legs spastic, there were present exaggerated knee-jerks and ankle-clonus. Ankle-clonus was more marked on the right side. There was also a slight scoliosis. The gait was spastic, there was no involvement of the bladder or bowels and no trophic lesions.

There was now noted a marked anesthesia on the outer aspect of the left arm and left forearm. The thermal sense was also involved in this area; the patient made repeated mistakes when tested for hot and cold. No sensory losses were noted elsewhere. An examination of the eye at this time revealed the pupillary reactions to be normal. No view of the fundus of the left eye could be obtained because of opacity of the cornea. In the right eye the fundus was normal. An examination of the urine was negative.

Little by little the spasticity and contracture of the arms and legs became more and more pronounced, until in March, 1898, she became unable to walk. The loss of the power to walk was exceedingly gradual. At various times she suffered from pain in the back and from severe pain in the arms and legs. The bowels also became markedly constipated and in addition there was difficulty in micturition; the patient was obliged to wait for quite a while before the bladder could be emptied and at times it became necessary to use the catheter. No trouble was noted with speech or deglutition. She complained of numbness of the right hand and of the right leg.

Examined in detail, July 29, 1898, she revealed the following: The patient is bed-ridden. She cannot move the right leg at all. She can flex and extend the toes of the right foot a little. She can barely lift the left leg from the bed, but can flex and extend the left leg feebly. She can abduct and adduct the left thigh a little. She can move the left foot and toes fairly well.

She cannot move the right arm at the shoulder at all, but can flex and extend the right arm feebly. There is almost no grip. She can move the left arm at the shoulder quite well and can flex and extend the left forearm quite well; also pronate and supinate the left forearm. There is no palsy of the face. The tongue is protruded straight and is not wasted. There is marked wasting of the intrinsic muscles of both hands, arms and forearms. There is no wasting of the legs or thighs. Insteps of both feet are very high. Patellar jerk is very marked on both sides. Patellar clonus is present on both sides. Ankle-clonus is marked on the right side, slight on the left. Plantar jerk present on both sides.

The sensory losses were now found to be more marked:—

*Tactile* sensation is abolished over the whole left upper extremity. There is no sensory loss over the right upper extremity except some impairment of sensation over the dorsal surface of the hand and fingers and a doubtful impairment over the palmar surfaces of the fingers. There is complete tactile loss over the left side of the chest, covering an area bounded above by the clavicle, below by the sixth rib, laterally by the middle line of the body. In the lower extremities, sensation is intact. An area of tactile anesthesia is noted, about two inches in diameter, extending downward and inward from the right nipple. There is tactile loss also on the left side of the neck; the face and ear, however, are not involved. On the upper portion of the back, on the left side, there is a complete tactile loss over an area extending from the junction of the neck and trunk above, downward to about two inches below the angle of the scapula and from the spine to the anesthetic area on the left side of the chest with which it merges.

The *pain* sense also reveals marked anomalies. There is complete loss to pain over the whole of the left upper arm, posterior surface of the left forearm and on the back of the left hand in an area corresponding to the first, second and third metacarpal bones and their respective fingers. There is a partial loss of the pain sense over the anterior surface of the left forearm, over the dorsal and palmar aspects of the hand in the region corresponding to the fourth and fifth metacarpal bones and over their respective fingers. There is complete loss of the pain sense over the left chest covering an area bounded above by the first interspace, below by the fifth interspace and internally by the middle line of the body. The pain sense was intact over the right side of the chest, the right upper extremity and both lower extremities. On the upper portion of the back, on the left side, there is complete loss of the pain sense over an area extending from the spine of the scapula down to the level of its angle; it does not extend entirely to the spinal column and laterally merges with the area of pain loss on the left side of the trunk. Partial loss of pain sense extends from the posterior border of the scapula over to the spine and downward as far as the limits of the area for tactile loss.

The *thermal* sense is abolished entirely over the left arm and posterior part of forearms, also on dorsal aspect of hand over first, second and third metacarpal bones and their respective fingers. It is almost entirely abolished over the area for loss of pain on the left chest; here hot and cold are confused, more usually cold being mistaken for hot. Over the anterior surface of the left forearm, there is a partial loss and confusion. Over the fourth and fifth metacarpal bones and their respective fingers, of the left hand, both on the dorsal and palmar aspect, there is some impairment, but hot and cold are usually called correctly. Over the posterior portion of the trunk, the thermal loss practically follows that of pain, but in addition includes the lower half of the ear and margin of the face about the angle of the jaw. It also extends toward the spine to a line about half way between the border of the scapula and the spine. From this line over to the spine, hot and cold can be distinguished. From one-half to one inch below the angle of the scapula down to the limit of the area of tactile loss, there is a partial loss of thermal sensations. This corresponds to the area for partial painful loss. The thermal sense is intact over the whole of the left thigh and leg, also over the right thigh. From knee down, over the whole of the right leg, there is an impairment which seems to vary at different parts, but is most pronounced over the internal malleolus.

November 9, 1898. Patient's condition practically unchanged. Complains now of tenderness in the ankles in addition to weakness. States now that she has always suffered considerably from pain in the joints and that this pain was made worse on motion. Mental condition somewhat obtunded so that she may be in error with regard to the past history of pain.

Ophthalmoscopic examination at this time again gave a negative result.

November 25, 1898. Patient now complains of a sensation of constriction about the waist. There is also diffuse tenderness to pressure over the calves, thighs and both arms. This tenderness is quite pronounced.

On November 30, 1898, the patient attempted to get out of bed. She fell, striking her head, causing a wound of the scalp half an inch in length. It was dressed antiseptically and subsequently healed promptly.

From this time on her condition changed gradually and almost imperceptibly.

She was again carefully examined on February 28, 1900. The symptoms had been very slowly progressive and but little change had taken place since the previous examinations. The following note was made:

Muscular wasting is now somewhat more pronounced. She cannot move the right arm at the shoulder. She can move it to a slight extent and flex it at the elbow. She can neither flex nor extend the fingers which are held in a position of semiflexion. The muscles of the arm, forearm and hand are all somewhat atrophied. The biceps jerk is increased and the muscle jerks of the forearm are increased. Sensation is quite well preserved in the right arm. She cannot move the left arm at the shoulder. She can flex and extend the left elbow, flex and extend the left fingers, but all the movements are weak. There is slight wasting of the muscles of the left hand and left forearm. The left biceps jerk is absent; the muscle jerk is slight. There is anesthesia of the left arm and left chest, the areas corresponding closely with those determined at previous examinations.

Both legs are edematous. Left leg can be slightly extended at knee, ankle and toes. Right leg can be flexed at knee very slightly, almost no movement in hip of left side, none in right hip.

Sensation to touch preserved in both legs and there is some hyperesthesia. There is no wasting of the legs. She localizes touch well in legs and feet. No fibrillary twitchings. She confuses heat and cold on both legs, sometimes answering correctly; at times wrongly. Edema of the feet.

Both knee-jerks are exaggerated. On the left side distinct ankle-clonus, also on right. Stroking either sole causes flexion of toes. No extension. Achilles jerk preserved on either side, but slight. Striking the sole causes flexion of entire foot. On the abdomen at right side, sensation to touch normal; on the left side, normal up to line of ribs. Heat and cold are felt perfectly well on abdomen. Marked constipation. Good control of the bladder. Occasionally dribbling.

Abdomen shows a freely movable irregular tumor, probably feces. There is no deformity of spine.

May 3, 1900. Patient is growing weaker. Diuretics have lessened the edema of lower extremities. Patient is very restless at night.

October 1, 1900. Patient is in very poor condition this morning. For past few days has had severe diarrhea with some blood in the stools; also considerable pain and bearing down; is very weak.

October 2, 1900. Has seemed much worse today. Pulse cannot be felt at the wrist and patient bathed in a cold sweat, with sub-normal temperature. During greater part of day has been unable to swallow any medicine or nourishment.

She died October 3, 1900.

The urine had been repeatedly examined during life and always with a negative result except with an occasional low specific gravity. Examination of the sputum had also yielded a negative result.

The body was removed from the hospital before an autopsy could be held. The resident physician, Dr. E. T. Robinson, followed the body to a distant part of the city and under discouraging circumstances succeeded in securing the spinal cord. Unfortunately no general post-mortem could be held. Microscopical examination revealed the following changes in the cord.

Sections from the upper part of the cervical swelling. The central portion of the spinal cord is much disintegrated so that the tissue in some parts has entirely disappeared; in other areas the tissue is very loose. This area of softening extends into the lateral column of each side, but more into the left than into the right lateral column. The crossed pyramidal and direct cerebellar tracts are degenerated. A glassy homogeneous infiltration in patches is seen in the central region of the cord, in the posterior columns, and in the lateral columns, especially in the left lateral column. This infiltration in some areas has replaced the normal tissue of the cord; in others it has penetrated between the nerve fibers leaving them more or less widely separated from one another and embedded in a homogeneous glassy matrix. Some much swollen axis cylinders are seen here and there in this infiltration. The walls of many of the intramedullary blood vessels are much thickened and have undergone a hyaloid change and in some more than one lumen is found. Disseminated sclerotic areas are found in different parts of the transverse section and not always in association with thickened blood vessels. Round-cell infiltration is not very distinct either in the cord or pia, although here and there a little infiltration may be seen about a vessel. Many of the nerve cell-bodies of the anterior horns, stained by ammonium carmine, are atrophied and with few or no dendritic processes. The anterior and posterior spinal roots appear to be normal, although the posterior possibly may be slightly degenerated. This, however, is very doubtful. Sections of the spinal cord above the upper part of the cervical swelling were not obtained. Sections from the middle part of the medulla oblongata are normal, except that the anterior pyramids were destroyed when the spinal cord was removed at the necropsy.

Sections of the cervical region taken from lower levels than those described above, present a similar picture. The nerve cell-bodies of the anterior horns, especially of the left horn, are very distinctly degenerated. Here and there a slight round-cell infiltration, about an intramedullary blood vessel, is found. The homogeneous infiltration of the cord—very pronounced at this level also—stains a light chocolate color by the Weigert's hematoxylin method and pale pink by ammonium carmine.

Sections from about the eighth cervical region are very similar to those described from higher levels, although the alteration, except in the crossed pyramidal tracts, is less intense. Here and there a small recent hemorrhage is seen within the spinal cord. Sections from this level stained by the Marchi method do not show any recent degeneration.

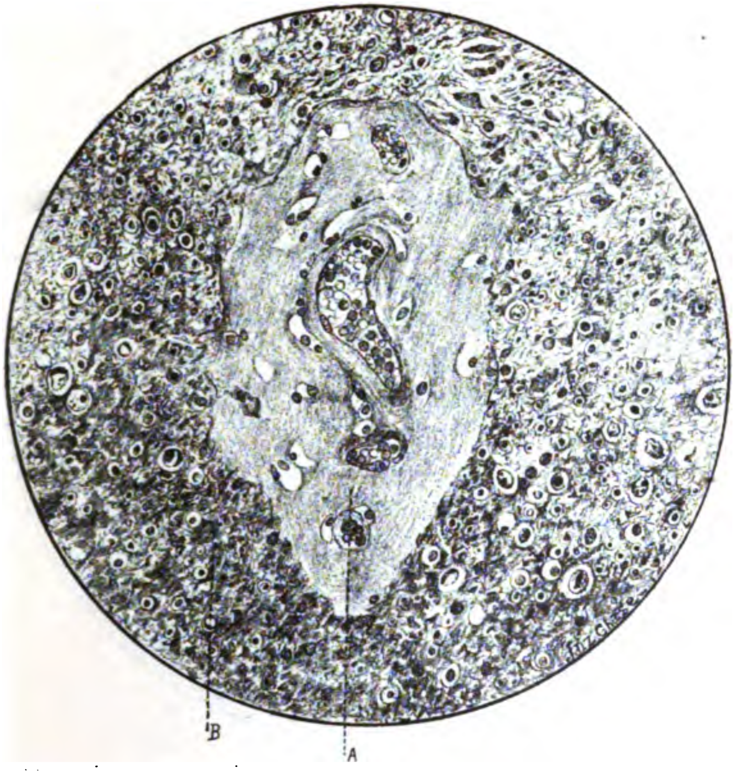

Fig. 1. Section from the upper portion of the cervical region. A, Blood vessel showing colloid-like degeneration of its walls. Three or four lumina are found within this vessel. B, Sclerotic portion surrounding degenerated blood vessel.

The degeneration of the crossed pyramidal tracts extends through the thoracic region into the lumbar.

The microscopical work was done by Dr. Wm. G. Spiller at the Pepper Clinical Laboratory.

It is to be regretted that the portion of the spinal cord above the cervical swelling was not obtained, but the findings are sufficient to explain the symptoms. Throughout the cervical swelling many of the blood vessels have undergone a hyaloid change, and a glassy infiltration, resembling that described by Obersteiner as colloid infiltration, has occurred, separating and destroying the normal tissue of the cord and causing softening of the cord and secondary degeneration.

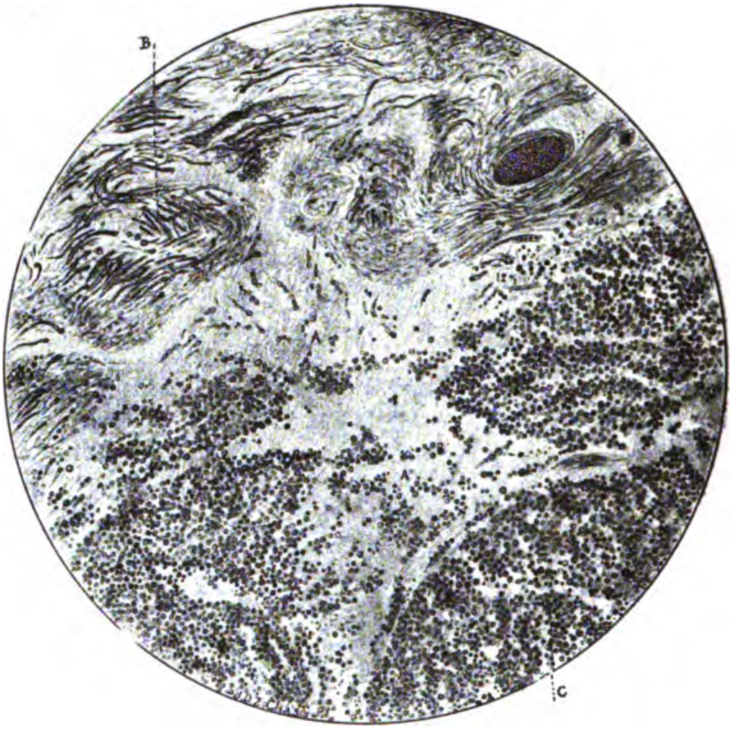

Fig. 2. Section from about the eighth cervical region. A, Homogeneous infiltration, staining light chocolate color by the Weigert hematoxylin method. B, Posterior horn and posterior commissure infiltrated by the homogeneous exudate. C, Posterior columns.

The only reference bearing upon this case, of which I have any knowledge, is that of Alzheimer[2], who describes two cases of colloid degeneration of the vessels of the brain. In one of these the symptoms were those of paresis, and Alzheimer regarded the case as one of paresis complicated by colloid disease of the blood vessels. In the second, the symptoms were those of brain tumor. The patient had during life choked disc, left-sided paralysis, convulsive attacks limited almost exclusively to the left side of the body, and headache. Numerous foci of softening and degeneration were found in the right hemisphere, in the right basal ganglia and in the right crus, together with colloid disease of the vessels.

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[2] Archiv f. Psychiatrie, Volume 30, page 18.

In the case here presented, a large focus of softening and infiltration was present in the upper cervical portion of the cord, besides there were areas of softening and infiltration present in various other portions as described. Colloid disease of the blood vessels of the central nervous system, presenting either diffuse or focal lesions, must be regarded as excessively rare and the case before us is unique. During life no definite diagnosis had been made. Early in the case syringomyelia and later on tumor of the cord seemed probable.

# ARTERIOSCLEROSIS OF THE SPINAL CORD.[1]

By William Hirsch, M.D., New York.

It is a well known fact that arteriosclerosis is a condition which may occur in many parts of the body; in the vessels of the muscular apparatus as well as in those of the internal organs. Heart, lungs, stomach, liver, kidneys, *etc.*, may become the principal seat of this affection, and many different types of the disease have been described accordingly.

The arteries of the central nervous system are not exempt from this affection, but may on the contrary form the starting point, or perhaps the only seat of the disease. Sclerosis of the cerebral arteries is a comparatively frequent occurrence, and its clinical manifestations, the physical and mental symptoms, are sufficiently known to lead as a rule to a correct diagnosis *intra vitam* without difficulty.

Arteriosclerosis of the spinal cord, however, is a condition which is only rarely diagnosed. The literature on this subject is extremely scanty; most textbooks not even mentioning it. Having been especially interested in this subject for several years past, I have collected a number of cases in which arteriosclerosis of the spinal cord formed the principal and sometimes the only feature of the disease.

As to the etiology of arteriosclerosis in general a great many conditions have been mentioned by various authors. Infectious diseases, chronic intoxications, abnormalities in metabolism, heredity, old age, *etc.,* were held responsible for the manifold types of arteriosclerosis. It is not my intention to discuss these various factors as to their relative value in the production of the disease. There is, however, one point which seems to me of special importance in connection with this subject. In spite of the many conditions which are considered of etiological significance, there remains a comparatively large number of cases in which arteriosclerosis can not be attributed to any real cause, so that we must assume a primary tendency on the part of the vascular apparatus to undergo degenerative changes. It is true, heredity has been found to play a certain rôle in this class of cases, but what else does that mean than that the individual is endowed with abnormal blood vessels *ab ovo*. In fact arteriosclerosis has been observed in infants only a few months old. I have seen sclerosis of the peripheral arteries and those of the heart in a number of young men between twenty and twenty-five years of age. While arteriosclerosis is extremely common in old age, we have to deal here with premature changes of the blood vessels, due to a congenital abnormality of the circulatory apparatus. This then accounts for the manifold idiopathic diseases of the internal organs and the central nervous system based on degenerative lesions of the arteries.

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[1] Read at the annual meeting of the American Neurological Association, June 5, 6, and 7, 1902.

In order to obtain as pure a clinical picture as possible of arteriosclerosis of the spinal cord, I have carefully eliminated all those cases in which even a suspicion of syphilis had to be maintained. Most cases which I have collected belong therefore in that category of idiopathic abnormality of the blood vessels.

Considering the anatomical basis of the disease it is evident that a sharply defined clinical picture cannot be given. It goes without saying that any artery or any group of arteries may become the principal seat of the lesion, and that according to the localization and intensity of the affection the symptoms will vary. Just as the clinical picture of spinal syphilis is characterized by the atypical complex of symptoms, so is arteriosclerosis of the cord a disease which may offer a great variety of clinical manifestations. In spite of this, however, there are certain clinical phenomena which will enable us to recognize this morbid condition of the spinal cord during life, and to differentiate it from other diseases of this organ.

One characteristic feature of spinal arteriosclerosis is the fact that the cells of the anterior half of the cord are much more apt to become affected than those of the posterior part. This is probably due to the peculiarity of the blood supply of this organ. The anterior spinal arteries are much less numerous than the posterior ones, and while the anterior arteries anastomose in such a way as to form one longitudinal vessel running over the entire anterior surface of the cord, the anastomosis of the posterior arteries produces six longitudinal vessels running parallel all the way down from the upper part of the cord to the conus. This abundant anastomosis of the blood vessels immediately before their entrance into the substance of the cord may afford a greater safeguard against an increased blood pressure, and in case of occlusion of one of the smaller arteries the anastomosis allows a ready blood supply from other sources. Of the anterior half of the cord it is again the lower part which becomes more frequently the seat of the disease than the upper part. The cause for this may lie in the fact that here the blood supply is still more direct than in the cervical and dorsal regions. The one artery which accompanies the sciatic plexus, the arteria magna, supplies the anterior gray matter of the entire lumbar and sacral cord, while in the upper part the spinal arteries are more numerous. The arteria magna has the largest caliber of all the spinal arteries, and the blood passes more directly from this vessel into the substance of the cord than at any other place. The arterioles of the anterior horns in the lumbar and sacral region have therefore less protection against an increased blood pressure than any other part of the spinal cord.

As a consequence of this condition we find in arteriosclerosis of the cord motor and trophic disturbances much more frequently than sensory symptoms, the latter in fact being rather rare in this affection. The existing manifestations again are more common and more marked in the lower than in the upper parts of the body.

The motor symptoms consist of gradually increasing weakness, characterized by fatigue after moderate exertion such as standing and walking, combined with a slight tremor of the head and of the upper and lower extremities. The gross muscular power gradually diminishes more and more, the gait becomes dragging and difficult, until finally, in the more severe cases, it ends in absolute inability to walk. There is, however, no atrophy as a rule, and the muscular tonus retains its normal condition up to a late period of the disease. The patellar reflexes may be exaggerated at first, then become sluggish, and may be lost entirely in the later stages. A slight tremor of the lower extremities may cause a moderate swaying of the body in the standing position, which, however, is different from the typical Romberg symptom. These may be the only symptoms of the disease for a long time; there are no sensory disturbances. Perception to outer stimuli remains unimpaired, and there is no pain. The reflexes of the bladder and rectum are normal. Sooner or later the other organs become affected in a similar way, and the patient may die from a cerebral hemorrhage or an intercurrent disease. This clinical picture is so often found in old age that it has frequently been considered a normal condition of this period of life. Unsteadiness of upper and lower extremities and gradually increasing impairment of gait and muscular power seem so common in old people, that one is often inclined not to look upon these phenomena as a morbid condition at all. But there are undoubtedly a great many old people who do not manifest any of these symptoms, and on the other hand, we not infrequently meet with these symptoms at an early period of life, so that it seems hardly justifiable to consider this condition a physiological process even among the very old.

Trophic disturbances manifest themselves in malnutrition of the skin, and of the mucous membranes, bones and joints. Glossy skin is a frequent symptom in these cases; the nails may become brittle, or may offer the conditions of onychogryphosis or alopecia unguium. There may be a tendency on the part of the skin to undergo ulceration and gangrene. A certain percentage of those cases known as erythromelalgia and Raynaud's disease are undoubtedly due to lesions in the trophic centers of the cord. The long bones may suffer from malnutrition, and possess a tendency to undergo spontaneous fracture. Certain forms of joint disease arthropathia and hydrops articulorum intermittens are found in this condition.

It is true that it is very difficult and often impossible to decide in individual cases whether these trophic disturbances are really due to lesions of the cord or to morbid changes in the peripheral arteries, especially as both conditions may occur simultaneously. There is, however, a considerable number of cases in which the peripheral vessels were found in perfectly normal condition, and where the cause had necessarily to be attributed to morbid changes in the trophic centers.

The diagnosis of spinal arteriosclerosis may sometimes offer considerable difficulty, especially when it occurs in younger individuals, and when its clinical manifestations resemble those of the well known systemic diseases. Although, as mentioned before, sensory symptoms are comparatively rare in arteriosclerosis, they may nevertheless occur. In the first place they may be produced by complicating peripheral lesions, and secondly, it is of course not impossible for the posterior part of the cord to be involved also. The clinical aspect of cases of this kind may closely resemble those of tabes. Here it is the history of the case, the absence of syphilis, arteriosclerosis in other parts of the body, and the condition of the pupils, which may be of value in the differential diagnosis. The Argyll-Robertson pupil does not belong to arteriosclerosis. If, on the other hand, the case is complicated by sclerosis of the cerebral arteries, we may have a difference in size of the pupils, or an atrophy of the optic nerve, leading to complete blindness. This latter condition, which is often considered one of so-called idiopathic optic atrophy, is by no means rare in cerebral arteriosclerosis, and can be explained by purely mechanical pressure. The optic nerve and the ophthalmic artery pass through the optic foramen in a common sheath. There are cases on record where the autopsy showed a marked thickening of the artery at this place, in consequence of which the nerves were compressed and caused to atrophy. If furthermore arteriosclerosis occurs at the posterior half of the lower part of the cord, it may through the production of proliferating interstitial tissue cause a secondary degeneration of the posterior columns, and in this way produce conditions, which even in anatomical respects, resemble those of tabes. It is possible that this fact accounts for those cases which are mentioned in all statistics as a small percentage of tabes without syphilis.

Progressive muscular atrophy, amyotrophic lateral sclerosis, and multiple sclerosis, will offer much less difficulty in their differentiation from arteriosclerosis. There is on the other hand spinal syphilis which may in its clinical features resemble arteriosclerosis so closely that the differential diagnosis becomes extremely difficult. Most cases of spinal syphilis take their starting point in the meninges, and as a consequence of this produce more general manifestations and especially root symptoms. If the gray substance becomes affected primarily the symptoms may resemble those of arteriosclerosis. Here it is the clinical course which may help us to differentiate between the two conditions. Syphilis is apt to produce acute attacks with sudden remissions and exacerbations. The clinical picture is apt to undergo manifold changes and fluctuations, while arteriosclerosis is a slowly but steadily progressing disease, without many changes or irregularities in its clinical course.

Arteriosclerosis hardly ever remains confined to one single organ any great length of time. It may do so at the beginning of the disease, or the symptoms of the one or the other organ may stand out more prominently during a longer period, but sooner or later it will become manifest that arteriosclerosis is a general disease, and that its confinement to one particular part of the body is seeming rather than real. Therefore arteriosclerosis of the spinal cord does not constitute a *morbus sui generis,* but forms a period of a general disease, of which it is true the spinal symptoms may for a long time stand in the foreground, or may even form the principal features throughout the disease.

In the following cases the diagnosis of arteriosclerosis was made purely from the spinal symptoms at a time when the other organs were in a fairly good condition. During the later course the symptoms became more general in most cases, other parts of the body, especially the brain, becoming affected. As mentioned before, only those cases were selected in which syphilis could be excluded with certainty, so that this disease would not come in consideration even as an etiological factor.

*Case* 1. Mr. G., fifty-two years old. Merchant. No heredity. No syphilis. Wife has four healthy children. No miscarriages. Use of alcohol and nicotine very moderate. No infectious disease except a few slight attacks of malaria during the last few years and scarlet fever in childhood. Patient consulted me for the first time in May, 1899. Complained about gradual loss of muscular power in his lower extremities. Since two years his gait had become more and more impaired. Besides this he complained of dizziness especially in bending down. Examination revealed perfectly normal condition of the cranial nerves. Pupillary reflexes and fundus normal. No atrophies. No change in muscular tonus. Sensation perfectly normal all over. Patellar reflexes both missing. Radial arteries very hard. Urine normal. Heart and other internal organs in good condition. Patient walks like a man of ninety years. The body is slightly bent forward, and the feet are slowly dragged, without lifting them from the floor. After walking for a few minutes in this manner he has to sit down on account of great fatigue. Standing for any length of time is equally difficult. There is a slight tremor of both upper and lower extremities. The mental condition is perfectly normal. Memory for the past as well as for recent events excellent. Intelligence unimpaired. No morbid emotions; speech without any disturbance. Writing somewhat unsteady on account of the tremor. When asked to bend his head down and raise it suddenly he became so dizzy that he had to be supported in order to prevent him from falling. The diagnosis of arteriosclerosis of the spinal cord was made. Patient remained in this condition for about two years; then the symptoms became still more marked, so that he was hardly able to walk at all. Two months ago cerebral hemorrhage and death.

The absence of the patellar reflexes in this case combined with the impaired gait and the motor disturbances due to tremor and dizziness resembling, to a certain extent, a moderate degree of ataxia, had led to the diagnosis of tabes, which, however, I rejected positively.

*Case* 2. Mr. N., sixty-four years old. Merchant. Heredity, alcohol, nicotine, syphilis and other infectious diseases negative. I saw patient for the first time in October, 1899, in consultation with his family physician, from whom I obtained the following history. He was always a healthy man up to a few years ago when he noticed a slight weakness in his lower extremities coming on very gradually. About eight months previous to the time when I saw him, he fell in the street and sustained a fracture of the left femur, complicated by a complete tearing of the tendon of the quadriceps. Patient could not remember that he had slipped in the street nor that his fall was due to dizziness or vertigo. All he knew was that his limb gave way and that he fell. He was treated surgically and made a complete recovery, so that he could walk about as well as he did before the accident. The weakness of the lower extremities continued and increased gradually. About five months later he had another accident in the street in spite of his extreme care in walking. This time he fractured the tibia of the left leg. He was absolutely sure that he had not slipped, and that no disturbance of consciousness could account for his fall. The examination revealed an absence of both patellar and both tendo-Achillis reflexes. No other symptoms on the part of the nervous system. Both radial arteries were sclerotic and the second heart sound was very much accentuated. The arteries of the lower extremities as far as they could be examined showed no particular change. The diagnosis of spinal arteriosclerosis was made, and the patient treated accordingly. Since that time he has remained in good health with the exception of the slowly progressing weakness of his lower extremities. There was never any pain in any part of the body.

*Case* 3. Mrs. B., forty-eight years old. Father died of apoplexy when fifty years old. One brother has heart disease; one sister highly nervous. No syphilis. Scarlet fever and diphtheria in childhood. Patient came to my office in November, 1900, with the following complaints: Since about a year she noticed a gradually increasing weakness of the lower extremities which manifested itself mostly in the standing position. Patient in fact claims to be unable to stand still. If not sitting she must move about. She experiences great difficulty in getting up from a chair and in climbing stairs. Walking produces fatigue very easily. She complains of a dull aching pain in the back and in the lower extremities, especially after any exertion. Occasional attacks of headache and vertigo. Patient is a very stout woman, weighing 212 pounds. Objective examination reveals no symptoms on the part of the nervous system, except patellar reflex on the right side missing, on the left side very much diminished. Tremor of upper and lower extremities. No atrophies. Radial and temporal arteries highly sclerotic. The right radial artery feels like a cord of iron. On both lower extremities large areas of varicose veins. On the anterior surface of the left leg an ulcer of the size of the palm of the hand is very sluggish in healing in spite of careful treatment. Systolic murmur of the heart and accentuated second sound. No enlargement. Internal organs otherwise normal. Urine contains no albumin nor sugar. Mental condition is in perfect order. The inability to stand is due to the combination of weakness and tremor of the lower extremities with the heavy weight of the body which makes walking easier than standing.

*Case* 4. M. C., forty-five years old. Tailor by occupation. Mother died from apoplexy. One brother has a spinal disease. Two sisters both nervous. No syphilis or other infectious diseases. Moderate use of alcohol and nicotine. Since several years weakness of lower extremities and pain on exertion. Nervous system shows no objective signs except absence of patellar reflexes. Sclerosis of radial and temporal arteries. Slight enlargement of the liver. No changes in the other organs. Urine normal.

*Case* 5. S. L., fifty-two years old. Peddler. Father died from brain disease. Three sisters and four brothers are all nervous. No syphilis or other infectious diseases. Admits considerable use of alcohol and nicotine. Patient complains about weakness and dull pain in upper and lower extremities. No shooting pains. Occasional headache and dizziness. Examination showed slight difference of pupils, the right being larger than the left one. No other symptoms on the part of the cranial nerves. Pupillary reflexes normal. Patellar reflexes exaggerated. Marked tremor of the tongue, upper and lower extremities. Sensation normal. Gross muscular power very weak. No Romberg; no atrophies. Nails are brittle. Multiple ulcerations at the tips of the fingers. One ulcer on the sole of the foot of the size of a silver dollar, looking like a typical mal perforant. Radial and temporal arteries highly sclerotic. The right radial artery can be felt as a hard string up to the middle of the forearm. Second heart sound accentuated. Other internal organs normal. No albumin; no sugar.

In all these cases the spinal symptoms stood in the foreground of the disease, although an arteriosclerosis could be demonstrated also in other parts of the body. In several instances the complex of symptoms resembled that of tabes just as cases of cerebral arteriosclerosis may resemble general paresis.

The following group represents a set of cases in which the spinal symptoms are combined with cerebral manifestations.

*Case* 6. Mr. H. S., sixty-five years old. Merchant. No heredity. No alcohol. No nicotine. Syphilis could be excluded with certainty. No other infectious diseases. I saw the patient at first in November, 1898, in consultation with his family physician, from whom I obtained the following history. He was always in perfect health up to two years ago, when the sight of the right eye began to fail. An oculist, who was consulted at that time, diagnosed a beginning atrophy of the optic nerve, for which no reason whatsoever could be detected, so that it was considered a case of so-called idiopathic atrophy. This process advanced slowly but steadily, and about half a year later the same condition took place in the other eye. At the same time the patient experienced some difficulty in walking, which also increased gradually. There was never any pain in the upper or lower extremities. When I saw the patient the atrophy of the optic nerves was advanced so far that he could only distinguish between light and dark. The pupils did not react even to the strongest stimuli of light. There were no symptoms on the part of the other cranial nerves. Patellar and tendo-Achillis reflexes were absent. No Babinski phenomenon. The muscular power of both upper and lower extremities was very weak and there was a fine tremor in hands and feet. Patient could walk only a few steps, and with great difficulty. Sensation was perfectly normal. The heart sounds were somewhat dull, but there was no murmur and no abnormality on percussion. The lungs were slightly emphysematous and the internal organs otherwise normal. No albumin nor sugar. Radial and temporal arteries showed no particular change. The mental condition was perfectly normal. I diagnosed the case as one of cerebrospinal arteriosclerosis, explaining the optic atrophy by the condition mentioned before, *i.e.*, by pressure of the enlarged and rigid ophthalmic artery on the nerve at its passage through the optic foramen. The weakness and the tremor of upper and lower extremities continued to increase gradually without being complicated by any sensory symptoms. During the following summer, while patient was in the country, he developed suddenly very marked mental disturbances. When I saw him he was in a state of great excitement, screaming and scolding. He had delusions of persecution and hallucinations of sight and hearing. The mental condition during the following weeks offered the typical picture of a case of dysphrenia, which is mainly characterized by the changeability of symptoms and lucid intervals. At times he was wild with excitement and at other times perfectly quiet. Now he was deeply depressed and melancholy, and then again gay and jolly. Here and there he had isolated delusions and hallucinations. All these morbid conditions were interrupted now and then by lucid intervals, during which his mental condition appeared perfectly normal. This special form of psychosis was in accordance with the diagnosis of arteriosclerosis, the symptoms being those of dysphrenia arteriosclerotica.

During the following year the lower extremities grew so weak that the patient became entirely unable to stand or walk, although there were no true paralysis, atrophies, or contractures. The mental condition kept on changing according to the nature of the disease. During the summer of 1901 patient had a hemorrhage in the internal capsule of the left hemisphere with right-sided hemiplegia and aphasia, which, however, receded after a few weeks, so that the condition became about the same as it was before, except for an increased weakness of the right arm. During the winter of 1901-1902 he had several attacks of edema of the lungs, which he also survived. Recently he got an edema of the hands and feet, and is in an extremely weak condition. The urine never contained any albumin.

Here we have a case with loss of patellar and pupillar reflexes; blindness, inability to walk and mental disturbances. How closely this complex of symptoms resembles those of a case of tabes, combined with general paresis, and still how different are both conditions in their pathogenesis. If this patient were about fifteen years younger the diagnosis of general paresis might have been very tempting, and still there is no reason why this very condition should not occur in younger men. I have no doubt that a certain percentage of cases which are reported in statistics as tabes and general paresis belong in this category, and that this, as said before, may account for the tabes and paresis cases without syphilis.

*Case 7.* Mr. A. T., forty-four years old. Merchant. Mother died from brain disease. One other brother died from apoplexy. Two sisters both "very nervous." No alcohol. Very moderate smokers. No syphilis and no other infectious diseases. When

I saw patient in October, 1900, in consultation with his family physician, he complained of general weakness, especially in the lower extremities. While in earlier years he was a good sportsman, he was unable now to run or even walk for any length of time. Standing was especially difficult. After the least exertion he experienced extreme fatigue and dull pain in his back and extremities. This condition came on very gradually during the last few years. Of late he was also troubled with a number of mental disturbances. At times he was depressed and suffered from loss of ambition and energy. Besides this, he complained of certain morbid fears and impulses, and of imperative ideas. Whenever he boarded a train of the elevated railroad or a ferryboat he experienced the impulse to jump down, and often asked the conductor to prevent him from doing so. Since recently he did not dare to take the elevated, but preferred the surface car for this reason. For days he was annoyed by certain thoughts and ideas, which he recognized as utterly absurd, but of which he could not rid himself. There were no objective symptoms on the part of the nervous system, except very sluggishly reacting patellar reflexes and a fine tremor of upper and lower extremities. Both radial and temporal arteries were as hard as iron cords, and considerably twisted. There were extensive varicose veins on both lower extremities and the ophthalmoscopical examination revealed sclerotic arteries of the retina. The outlines of the heart were normal, and there was no murmur, but the sounds were extremely intense. The urine contained traces of albumin, and the other organs were normal. I made the diagnosis of arteriosclerosis of the central nervous system. Half a year later patient died suddenly from cerebral hemorrhage.

*Case* 8. A. W., fifty-two years old. Tailor. Father died from apoplexy. No alcohol. Moderate use of tobacco. No syphilis. Has seven healthy children. No other infectious diseases. Patient consulted me in September, 1900, on account of weakness of the back and lower extremities, and of dull pain in these parts after any kind of exertion. Besides this, he complained of headaches, dizziness and ringing in the ears. These symptoms had come on gradually during the last two years. Up to that time he had always enjoyed good health and strength. There were no objective symptoms on the part of the nervous system, except a fine tremor of the hands, slight unsteadiness of the lower extremities, and somewhat exaggerated knee-jerks. Radial and temporal arteries were very hard. The heart sounds were very much accentuated, but there was no murmur or enlargement. Other internal organs normal. No albumin nor sugar. There were very large extensive varicose veins on both lower extremities. Nails were brittle; skin dry and badly nourished. The diagnosis of arteriosclerosis of the central nervous system with the complex of symptoms of Menière's disease, was made.

Patient's condition grew gradually worse; especially the spinal symptoms increased quite rapidly. After about six months patient was hardly able to walk. The patellar reflexes had become very slow and sluggish, but there was no atrophy and no pain except after exertion. After another six months patient developed mental symptoms consisting of depression and occasional maniacal outbursts. Now and then there were remissions in his psychical symptoms, lasting one or sometimes several days, so that the psychosis offered the clinical picture of a dysphrenia. Intelligence and memory remained unimpaired throughout the disease. In December, 1901, patient died from cerebral hemorrhage.

Autopsy: Edema of the brain. Pia can be removed from the brain without difficulty; extensive hemorrhage in the left internal capsule. Arteries of the brain and cord very sclerotic. Left Sylvian artery showed several white thickenings. Right Sylvian artery also considerably thickened throughout its course. Vertebral artery highly sclerotic. Brain and cord were hardened in five per cent. formalin. Sections were stained with Weigert, Nissl, Van Gieson and carmine. The anterior longitudinal artery of the cord was very much enlarged in diameter throughout its course; especially the adventitia seemed to be thickened considerably. The lumen of the vessel varied at the various levels. At some places it appeared somewhat contracted and packed with blood corpuscles, and at other places it was enlarged and irregularly shaped. The posterior longitudinal arteries showed similar conditions, with the exception that here the lumen of the vessels seemed not to be enlarged but rather diminished in size throughout. Both anterior and posterior spinal arteries showed considerable thickening of the walls. The arterioles of the gray matter of the cord appeared to be much thicker than normal. The lumen was often extremely small and sometimes seemed to be occluded entirely. At several places there were blood corpuscles on the outside of the vessel, and small groups in the substance of the gray matter, indicating minute hemorrhages. The motor cells in the anterior horns were normal in size and shape, but here and there the Nissl bodies appeared rather indistinct, and there was a very marked increase of pigment in the bodies of the cells. The cells of the posterior horns showed no morbid changes whatsoever, and the white substance was perfectly normal throughout the entire cord. The microscopical examination of the brain showed also general arteriosclerosis. A great many of the smaller arteries and arterioles were occluded, and several capillary hemorrhages had taken place into the substance of the brain.

All the foregoing cases represent the spinal type of arteriosclerosis sufficiently advanced to produce such anatomical changes in that organ that the diagnosis of an organic lesion could be made without difficulty. The question is now whether we would not be able to recognize this condition at an earlier period of the disease, at a time when no gross changes in the substance of the cord have yet taken place. There can be no doubt that every case of spinal arteriosclerosis must give some symptoms at such an early period, and that some cases may become stationary and never give evidence of an organic lesion. Naturally cases of this kind are as a rule put in the large indefinite category of neurasthenia. Although I do not claim to be able to lay down certain diagnostical rules, to separate these earlier or minor cases of spinal arteriosclerosis from purely functional nervous disorders, I want to point out a certain group of cases in which the diagnosis of beginning spinal arteriosclerosis at least suggests itself, considering the clinical manifestations of the well-marked cases described above.

The following case is only a representative of a frequent and well known form of disturbance:

*Case* 9. Mr. F. T., forty-three years old. Merchant. Father died from apoplexy. Mother and two brothers are nervous. No alcohol nor nicotine. No syphilis nor other infectious disease. Since a number of years patient complains about general fatigue, especially of the lower extremities, after the slightest exertion. His skin is dry and badly nourished. Nails are brittle. Circulation is sluggish. Patient complains constantly of cold feet. After a cold bath it takes him a long time to restore his circulation and to become warm. He suffers from sleeplessness. In the morning he feels extremely heavy and tired, and the eyes are bloodshot. The examination reveals no objective symptoms except rigid radial arteries.

This case represents, as said before, a type of patients who are generally classified among the neurasthenics, and who are by no means rare in the practice of any physician. If we compare his symptoms with those cases of well marked spinal arteriosclerosis, we must admit that a striking resemblance exists between them, and that at least the possibility of this case belonging in the same category cannot be denied. It is evident of what importance it would be, to recognize the real pathogenic origin of this class of cases, for not only scientific but also for practical therapeutic purposes.

So much, I think, can be said today, that a special type of arteriosclerosis exists, which has to be located in the spinal cord. It must be left to future investigations to advance our knowledge of this somewhat obscure process still more, and especially to throw more light on the origin of this morbid affection of the vascular system.

# THE MICROSCOPIC FINDINGS IN FOUR GASSERIAN GANGLIA REMOVED FOR TRIGEMINAL NEURALGIA.[1]

### WITH A RÉSUMÉ OF TWO PREVIOUSLY EXAMINED.

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In the *Annals of Surgery* for June, 1901, I reported the microscopic findings of the examination of two Gasserian ganglia removed by Dr. Bartlett for persistent and very severe trigeminal neuralgia. The conclusions reached then in respect to the pathology of this disease were tentative, and it was for the purpose of testing their correctness that a study of four additional Gasserian ganglia was made. I am indebted for them to Drs. Bartlett, Mudd, and Carson, the latter for two, and I wish here to express my thanks for the specimens as well as for the clinical notes of the cases from which they were removed.

The experience in regard to methods of sectioning, staining and general technic gained in the previous study were of great use in the present series, and it can be justly said that changes which might be due to accidental causes have been avoided as much as it is possible to do so with the technical methods at present at our disposal. The value of the findings in the present series is further increased by the fact that two of the ganglia were removed before any peripheral operation had been done on the branches arising from them. In this way the traumatic effects of stretching, resection, and forcible evulsion were not present to invalidate whatever positive findings in the nerve cells were obtained. In one of them, the one numbered 5, I have been able to use a technic for hardening, the most perfect that has as yet been devised. This method was employed and modified by Orr and Rows in studying the posterior root ganglia in dementia paralytica.

In the consideration of that ganglion, I shall refer to the technic more in detail. In the first ganglion studied the Marchi stain was very successful, and it showed beyond question that the effect of operation on the peripheral branches of a Gasserian ganglion is to be seen in the production of marked changes in the peripheral branches.

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[1] Read before the St. Louis Medical Science Club, November 11, 1902.

For the general considerations of the anatomy and of the literature of this subject I shall refer to the article previously alluded to. Since that was published, I am unable to find any additional data of any importance on the microscopical study of such ganglia, although they must have been removed a great many times. I shall describe in some detail the findings in each of these ganglia, numbering them 3, 5, 6, and 7; then I shall attempt to summarize them, with the view of bringing them into relation with the general pathology of trigeminal neuralgia. (The ganglion numbered 4 was omitted from this series on account of its imperfect condition.) I shall then lay before you, chiefly for your criticism, certain conclusions in regard to the pathology of trigeminal neuralgia, which the study of these six ganglia has seemed to justify.

In the paper previously referred to, certain conditions were laid down which should be always present before the findings in any given ganglion can be accepted without criticism. As I have had no reason to alter them, I shall state them again here. (1) A Gasserian ganglion, upon the peripheral branches of which surgical operations have previously been made, in the way of nerve stretching, resection, *etc.*, is unfit for pathological study, or rather, any conclusions drawn from the findings must be for the most part invalidated for the reason that the mechanical effects of the operation may cause an ascending neuritis, which might produce changes in the ganglion itself, in the cells, or in the periganglionic tissue. (2) A Gasserian ganglion, which is removed by morcellation, or is much torn or cut, cannot be regarded as a favorable object for microscopical study. (3) No conclusion in regard to the condition of the nerve cells is justified unless they have been studied by the Nissl method or its various modifications. Of course the ganglion must be examined in a good state of preservation.

In regard to the general technic, I may add that three of the ganglia were imbedded in paraffin and one in celloidin. All of them were cut transversely and parallel to their largest diameter, so that every section contains the whole body of the ganglion, and the cells stained in such a section were the total number present at that level. Complete serials were made in every case, and sections from different levels were stained, and only the most perfect were saved for study and description.

Ganglion No. 3. This ganglion was removed by Dr. H. G. Mudd from a patient about fifty years old, in 1901. In 1896 the infraorbital nerve was cut and pulled out at the infraorbital foramen, after being divided inside the orbit. The inframaxillary branch was cut at the mental foramen and the nerve evulsed. Cells: The cells toward the center of the ganglion are mostly normal, filling the cell spaces with almost no retraction. They take the stain however very irregularly, those staining most deeply with the methylene blue or thionin, *etc.*, show the greatest irregularity in form. The cells, in respect to their reaction to the stain, may be divided into two divisions: first, cells which are very nearly circular in outline, filling up the cellular spaces almost completely and staining very faintly, but otherwise normal in outline, nuclear position, and arrangement of Nissl bodies; second, cells which are very irregular in outline, smaller in size than the foregoing, showing marked retraction, and staining very intensely with the methylene blue stains and others of a like nature. These cells are fewer in number than the others, and most of the abnormal cells are found among them. In general they occupy a peripheral position in the ganglion body. They correspond in every way to similar groups of cells found in the first two ganglia studied, and seem analogous to a group of cells found by Head and Campbell in their study of the posterior spinal ganglia in herpes zoster. These writers supposed that such cells had to do chiefly with the function of pain, and therefore would show the most marked changes in a condition in which pain is the most prominent symptom. In a disease, such as neuralgia, where pain is the chiefest and most often the only symptom, changes in such cells might be expected to be present. These cells show almost every change which has come to be regarded as an evidence of the abnormal condition of a nerve cell. In most of them the changes seem to point rather to a disturbance of function than to a primary intracellular pathological process. I mean by that, that they show changes of outline and in the chromatic granules rather than in the position and character of the nucleus. In some few cells even the most profound alterations are present, such as migration or disappearance of the nucleus, total chromatolysis and cell disintegration. The pigment deposit in some of the cells is somewhat similar to that previously described in the ganglion numbered 2 in the former paper. This peculiar pigmentation is so unusual that it merits attention. Pigmentation in nerve cells is not commonly considered to be abnormal, that is, when it occupies the cell periphery, as is usually the case. The pigmentation found here is perinuclear. The nucleus is surrounded by a ring of pigment granules, varying considerably in size. The ring of pigment seems to lie above the nucleus, but the nucleolus is left free. In this specimen every tenth cell perhaps has such a perinuclear ring of pigment. It is to be noted that this curious pigment deposit is found mostly in the group of cells belonging to the first class, that is, the cells occupying the central part of the ganglion, which stain, as a rule, faintly and show otherwise little abnormality in outline or cell structure. Some cells show the usual peripheral deposit of pigment, to which no especial importance can be attached. Some of the sections, stained by Marchi for the study of degeneration in the peripheral branches, included cells from the body of the ganglion, because the cross sections of the roots were made close to the body of the ganglion. Some of the cells so included showed this perinuclear deposit, which stained black with osmic acid. Whether this is a proof of their fatty nature or not I am unable to say, but their appearance in such preparations is sufficiently striking to deserve mention.

Peripheral branches: In the study of the peripheral branches the Marchi and Van Gieson stains were used with good effect, especially the former. Portions of the three peripheral branches and also the sensory branch were cut both in cross section and longitudinally. In the first and second branches only very slight evidence of degeneration in the Marchi sense was observed. In the third branch marked degeneration was demonstrated. This consists chiefly in the usual clumps of degenerated myelin with swelling and knobbing of the axis cylinder. If you will remember that the third branch in this case had been forcibly evulsed some years ago, and that in all probability a slowly ascending neuritis resulted, these findings will be clear. Some of the sections from the sensory root show abnormal appearances by the Marchi stain.

It is rather difficult to explain this fact, because the changes are not characteristic enough to be designated as a neuritis. I am not inclined to believe that a neuritis of the sensory root exists of sufficient intensity to show itself by any microscopical method. If this were true, the good results obtained by the removal of the Gasserian ganglion would be hard to explain. It seems to me that what we have here is not a neuritis at all, but simply a destruction of the nerve fiber caused by the pressure of the forceps, for it must be remembered that in the modern operation, the sensory root is held tightly grasped by a locking forceps, while the ganglion body is being freed from its bed and while the peripheral branches are being cut. In the reports of the microscopical study of this subject, I have found one or two instances of neuritis of the sensory root, but they have not been studied carefully enough to make this point certain. It appears that the explanation above attempted is the fairer one at present. There was no definite alteration found in the blood vessels or in the connective tissue either surrounding the ganglion or lying between its cells.

Here and there throughout the ganglion free blood was found. The usual number of concentric bodies, the corpora amylacea, were present. The blood was no doubt produced by the operation. Summary: Cell changes, secondary in character, neuritis in the third branch, perinuclear pigmentation.

Ganglion No. 5. This specimen was removed by Dr. Bartlett from a woman about fifty-five years old. I think there had been no previous peripheral operation. This was a most perfect specimen. The case was of great interest clinically also for the reason that the patient developed, one or two days after the operation, a crossed paralysis, characteristic of a pontine lesion. This was caused, no doubt, by a slight hemorrhage into the pons, due to the tearing out of the sensory root. The paralysis cleared up within a week. I wish to say a few words concerning the method of hardening employed in this ganglion. It is a modification of Mann's method, and has for its object the avoidance of artifacts and the prevention of cell retraction. The authors claim for this technic that it presents an absolutely natural picture of the cells and that any retraction of the cells which is found must have existed before the specimen was hardened. This method was employed chiefly in the study of the posterior root ganglion, and for this reason is especially well adapted to the Gasserian ganglion. The similarity in anatomical structure between them has been previously pointed out in a former article. The method is as follows:

Fixing Solution: Picric acid, sat. sol.; sublimate, sat. sol.; formaldehyde. Equal parts. Three days.

Alcohol, beginning with forty per cent.

Thin celloidin.

Origanum oil, paraffin, equal parts, 12 hours, temperature, 45 to 50 deg.

Paraffin 12 hours.

The stain chiefly used was the Held toluidin blue, with a counterstain of erythrosin. This method gave satisfactory specimens.

Cells. The same irregularity that was noted in the previous specimen and the same division of cells were present. The cells lying in the periphery showed the same alterations in point of staining qualities and the same deviations from the normal. The centrally situated cells can, for the most part, be considered to be normal, with possibly a too faint staining reaction to be regarded as absolutely so. The smaller cells showed great irregularity in outline and in internal structure; some of them were so altered that they did not appear to be sensory cells at all. It is interesting to note the sharp contrast between the peripheral cells and those cells lying in the central part of the ganglion. The latter show no cell retraction at all, carrying out the contention of the authors alluded to before, while the peripheral cells are often so shrunken that the spaces between the cells and their pericellular limits were often larger than the cell-bodies themselves. There was an almost complete absence of the perinuclear variety of pigmentation. Whatever abnormality there was seemed to be limited to the smaller peripheral cells, which, on the whole, showed very much the same deviation from the normal as the cells in ganglion numbered three. No alteration was found in the blood vessels or in the interstitial tissue. Peripheral branches, as well as they could be studied by the Van Gieson stain, show no evidence of degeneration. The specimen contained a large number of concentric bodies, scattered irregularly throughout.

Summary: Beyond the abnormal appearance of a group of small cells lying in the periphery of the specimen, very little abnormality can be found. This was the most perfect ganglion that I have examined, so that it can be taken as the one which best represents the appearance of the Gasserian ganglion removed for severe trigeminal neuralgia.

Ganglion No. 6. This ganglion was removed by Dr. Carson from a patient about fifty-four years old, who had previously not been operated upon. The specimen was imbedded in paraffin and was not a very satisfactory preparation, chiefly on account of the difficulty in obtaining sections that were thin enough. The Held stain was chiefly used.

Cells: They show more deviation from the normal than in any of the preceding specimens. The cells show the most marked alterations in outline. Here, as in the two preceding ganglia, the division into two sorts of cells seems to be justified. The cell changes consist for the most part in greatly irregular outlines with marked retraction of the cell-body. In many cells the nucleus occupies a peripheral position, and sometimes is entirely absent. Such cells as these can be said to be definitely pathological. Very few cells seem to be normal, even the centrally lying cells, found so nearly perfect in the two preceding specimens, are here changed. There is much pigmentation of the cells; the perinuclear variety being very frequent, though scarcely as marked as in ganglion No. 3. The blood vessels and interstitial tissue appear normal.

Summary: Very definite and profound cell changes throughout the whole ganglion, irrespective of the position of the cells. The changes are so marked that they must be regarded as the seat of pathological changes in spite of the imperfection of the specimen from the microscopical point of view.

Ganglion No. 7. Removed by Dr. Carson from a patient upon whom no peripheral operation had been performed. The patient had been under treatment for a severe neuralgia for a period of ten years; during this time he had taken an immense amount of bromides and analgesics. The specimen was hardened in celloidin and stained by Nissl, thionin, and Van Gieson, *etc.* The specimen was much like ganglion No. 5 in general appearance and in stain reaction. Division of cells is the same as in the previous specimen. The changes consist in great irregularity of outline. The form of many of the cells is completely changed, from that of a round or oval body to an elongated or pyramidal one. The typical globular form of a sensory cell in the Gasserian ganglion is completely altered. The Nissl bodies are very irregular in their arrangement, the concentric form being seldom seen. Chromatolysis and vacuolization are seen to some extent, at least, in almost every cell. The cells themselves stain very irregularly; this causes a peculiar cell appearance, one part of the cell being deeply stained while another is almost without any stain at all, with absolutely no Nissl bodies to be made out. This peculiar intracellular stain reaction is not found in the cells of any other ganglion examined. In some of the cells lying deep in the ganglion there was a nuclear migration without any other cell change. This could be seen especially in the lightly stained cells. The number of abnormal cells in this ganglion is so large that they cannot be due to accidental causes. In the specimens are to be found very few normal cells, and the appearance of the ganglion as a whole is so different from the normal that one cannot escape from the conclusion that it is definitely the seat of pathological changes. There is no pigment deposit of importance and no alteration in the blood vessels or interstitial tissue.

The findings in these four ganglia and in the two previously reported might be summarized as follows: In all of them a certain number of distinctly abnormal cells have been found. This number is too large to be accounted for by the accidental effect of faulty technic, or through hardening or straining processes. The cell changes have been found mostly in the peripheral portions of the ganglia, and in those cells which are characterized by staining deeply, by elongated outline, and by their small volume as compared to the cells which lie in the deeper portion of the ganglia. The changes found in the cells vary all the way from a slight degree of chromatolysis to profound changes of total cell disintegration and nuclear migration. In three of the six ganglia perinuclear pigmentation was present in large numbers of cells. This is so different in position and character from the pigment found in many normal cells, that it must be, for the present at least, regarded as a deviation from the normal. In one of the ganglia a sclerosis of the periganglionic tissue with excessive connective tissue formation around and between the cells was found, which was so pronounced that its pathological significance is unquestioned. In some of the ganglia neuritic changes were found in the peripheral branches, but only in those cases in which a peripheral operation had previously been done. No certain changes in the blood vessels could be demonstrated. Concentric bodies or corpora amylacea were found in all the specimens, but they have no interest beyond their chemical or histological study.

In view of these findings, then, there is this question which confronts us: Have we a sufficient basis for the establishment of a pathology of trigeminal neuralgia, that is, for a pathology based upon anatomical variations from the normal? Before discussing this question, it is well perhaps to consider something about the data upon which we have to rely for an answer. If these six ganglia do not afford any basis for the answer to this question, six more or sixty more will certainly not. All of these ganglia were from cases of the most severe form of neuralgia; in three of them there had been no peripheral operation to complicate the microscopical findings, and all of them were removed cleanly and given to me in a fresh state, well adapted for histological study. In other words, accidental sources of error were removed as far as it is possible to do so at present, and the most approved technic was used in preparing them, and as far as I know nothing was omitted in the way of staining, hardening and fixing to give accurate preparations. It seems then a fair conclusion that, if trigeminal neuralgia depends upon disease of the Gasserian ganglion, these six ganglia should show sufficient anatomic changes to explain its chief symptom. If, on the other hand, trigeminal neuralgia does not arise from disturbance of the Gasserian ganglion, then the changes here described must have some other explanation. A further conclusion is forced upon us, and that is that if the Gasserian ganglion is the seat of trigeminal neuralgia and no changes in the ganglion sufficiently intense to explain the symptoms can be demonstrated, then either our methods of microscopical examination are at fault, or it is impossible to connect changes in the nerve cell with manifestations of abnormal cell activity, as is shown in this disease by the frequent attacks of the typical neuralgic pain. There is little doubt in my mind now that the principal seat of all trigeminal neuralgic symptoms is found in a diseased condition of the Gasserian ganglion. The proof of this, and I can see no way out of it, is found in the complete disappearance of symptoms when the ganglion itself is removed, or when the sensory root is divided, as has lately been done by Frazier at Spiller's suggestion. As soon as the ganglion is isolated, that is, as soon as its sensory pathway to the brain, where, of course, the conscious manifestation of pain is situated, is blocked, at that instant neuralgia of the quintus ceases. In the face of this anatomical proof, we must conclude that the causes which produce the attacks of pain must have their anatomical origin in the cells, the tissue, or the nerve fibers which compose the structure of the ganglion. Having now localized the only possible seat of the disease, anatomically considered, we may take up the other two questions. As far as the reliance on microscopic technic is concerned, it can safely be stated that we can rely upon its results when the technic is followed out with reasonable care and when enough material of the same sort is present as to overcome the natural sources of error. Six ganglia and many hundreds of sections seem to me sufficient to overcome this difficulty.

It seems fair then to say that whatever changes in the ganglia were present were shown by the methods employed with sufficient accuracy to make use of them towards an explanation of the disease. Of course it must be recognized that the limits of microscopical technic are of themselves sufficiently narrow to prevent any but a crude attempt at the explanation of diseased function. The last question to be considered is the ever present difficulty of connecting abnormal cell conditions anatomically with abnormal cell functions physiologically. This in truth is the crux of the whole question, and it is by no means limited to the problem here under discussion.

Given a nerve cell or cells in a Gasserian ganglion from a case of trigeminal neuralgia, having presumably to do with the function of pain, how can we in our own minds connect, let us say, the migration of their nuclei with the agonizing pain present in all such cases? This very point has been recently discussed by Schenck in his monograph on the physiological basis of the neurone theory. He especially emphasizes the fact that physiological processes may go on in the nervous system without their cells taking an active part in them. If this is true physiologically it must be true in the pathological sense also. In considering the chief symptom of neuralgia, that is, the recurrent attacks of pain, we must face the fact that our conception of the possible causes of pain are narrowed down to two, and perhaps to a single element. Briefly, we can comprehend mechanical causes for pain in the larger sense of the term, such as pressure, stretching, weight, *etc.*, and we can somewhat as readily comprehend certain chemical causes of pain, such as for example, the effect of an acid on an exposed nerve fiber. We are able to do this because we have experienced their effects. All other causes we cannot correctly reason about because we cannot directly experiment with them on ourselves. For example, if we try to think of any intracellular cause for pain, we must deal immediately with purely metaphysical conceptions. That there exists, or rather that there can exist, a purely parenchymatous cause of pain, is altogether possible, but at present, pathology, as we know it, does not seriously concern itself with it. There is little doubt that certain cases of trigeminal neuralgia are caused by the effects of mechanical pressure on the ganglion. There have been a few cases reported where tumors or traumatic bone lesions were found sufficiently near the ganglion body itself to exercise mechanical pressure upon it. Sclerosis of the ganglion itself may also be regarded as a cause acting mechanically. The deposit of connective tissue by pressure on the nerve fibers may well produce the attacks of pain which we so well know. But these extrinsic causes, acting mechanically, must be but a small factor in the causation of neuralgia, for their presence is a matter of great rarity in the published reports on the study of Gasserian ganglia removed for neuralgia. It is well, however, to remember that this is an occasional factor.

There are two other explanations remaining. The toxic nature of the disease and the intracellular origin of it. These might well be taken together because I think they can in a measure be brought into relation with the abnormal findings present in these six ganglia. I take them together for another reason also, because they both remain an unknown quantity, and it is only through their assumed effects that we speak of them. First of all I want to emphasize the fact that the cell changes which I have described are not the causes of neuralgia in the sense of a primary cell disease, but are either the effects of an abnormal or a greatly exaggerated cell activity. Whether these cells are primarily concerned in the production of pain is of course an undecided question, but it seems very probable that they are. These abnormal appearances in the six ganglia examined mean that the cells so affected have been over active for a long period of time, or that they have been abnormally active, so that gradually they have lost their anatomical integrity. They may be in a measure compared to the nerve cells in a state of intense fatigue, which Hodge has pictured in his experiments made at Clark University some time ago. In so far then we have the right to regard these ganglia as pathological, and every ganglion removed for the severe grade of neuralgia, for which alone the operation is justifiable, ought to show, if carefully examined, such large numbers of cells abnormal in this sense, that some account of them must be made.

As to the active causes leading to such pathological activity, we are mostly in the dark and any attempt at explanation must be purely speculative. There are two reasonable explanations: first, that there is some toxin circulating in the blood, which has a selective action on the cells of the Gasserian ganglion, usually on one side, and especially on a certain group of cells which may have to do with the function of pain. The nature of this toxin is absolutely unknown. Second, the cells themselves originate the toxin owing to some abnormal cell activity. That cells have this ability is proven by the recent studies on immunity. Possibly both of these factors are at work along with others of which we know as little. The results, after all, of this ganglion work in neuralgia, which at first sight are not very encouraging, are really of some importance, and I think that the line of inquiry which the study opens for us is well worth pursuing.

The following conclusions seem to be justified:

1. In six ganglia removed for trigeminal neuralgia, anatomic changes were found of sufficient importance to bring them in relation with the symptoms of this disease.

2. These changes in the cells do not point to them as the primary cause of neuralgia, but they may be taken to represent the effects of abnormal cell activity, the origin of which we are not able to point out.

3. The Gasserian ganglion is the anatomical seat of the chief symptom of trigeminal neuralgia, that is the attacks of neuralgic pain.

# Society Proceedings

## NEW YORK NEUROLOGICAL SOCIETY.

November 4, 1902.

The President, Dr. Joseph Collins, in the chair.

*Successful Laminectomy for Spinal Cord Tumor.*—Dr. Pearce Bailey presented a man who had been referred to in a communication to this Society made by Dr. McCosh. The operation had been done by Dr. McCosh in May, 1900. In December, 1898, when the patient was thirty-nine years of age, the illness began with sharp, shooting and intermittent pain in the leg. It extended from the sacral region down the posterior surface of the limb to the knee. He did not work from November, 1899, until he came under observation in May, 1900. At that time he was suffering intensely and could only walk with difficulty even with the aid of a cane. The pain affected the left lumbar region, the back and the front of the thigh, although previously it had been confined to the sciatic distribution. Examination showed an atrophy of the left leg with weakness and diminution of the knee-jerk on that side. There was very slight anesthesia over the area corresponding to the lower lumbar distribution, and also on the front of the thigh. There was some difficulty in passing urine. The anesthesia was considered very suggestive of tumor, involving as it did the anterior surface of the thigh. Laminectomy, involving the eleventh dorsal and down to the second lumbar vertebra, was done, and disclosed a bluish tumor which was removed. The result of the microscopical examination left it uncertain whether the tumor was a small-cell sarcoma or a mass of fibrous or granulation tissue, but the subsequent history pointed to the latter diagnosis as being correct. The man was able to continue at his usual occupation, which involved a good deal of muscular exertion, and experienced no trouble from the back, despite the laminectomy. Both knee-jerks were present now, and the anesthesia had disappeared from the front of the thigh.

*Osteosarcoma of the Spinal Column; Treatment with Coley's Fluid.*—Dr. Joseph Fraenkel presented a young man who had been well up to three years and a half ago, when he was struck in the back. For the next six months he was able to continue at work, but then the pain in the back became severe. When seen by Dr. Abrahamson a diagnosis of Potts' disease was made, and this was confirmed by Dr. Gibney, and the patient was put in a plaster of Paris jacket. About a year later there was total paraplegia. He was then admitted to the Montefiore Home. There was absolute motor paralysis of both lower extremities with absence of the tendon reflexes. There was an area of anesthesia about the size of a dollar in the anal region. There was no tenderness, and no external evidence of disease of the spinal column. Three or four months later a projection developed, which was most marked at the tenth dorsal vertebra. Exploratory puncture revealed no pus. Two weeks later a large swelling appeared on either side of the vertebral column. An exploratory operation showed the tumor to be solid, and a portion was excised for examination. Dr. Harlow Brooks reported the growth to be a highly vascular sarcoma. Injections of Coley's fluid were begun on February 13, 1902, though some improvement had been noted before this time. The treatment was continued up to May 15. At times the reactions were quite violent. After stopping the treatment the patient's general condition improved markedly, and the tumor diminished in size and became harder.

*A Case for Diagnosis.*—Dr. W. B. Noyes presented a man of twenty-six who fell three days previously from a building while at work. When seen the day before the meeting of the Society, he was walking on his tiptoes with the aid of crutches. Examination showed no sensory changes and no disturbance of bladder or rectum, and vision was normal. The reflexes were all increased. The case was presented for diagnosis. He looked upon it as one of hysteria, although he had thought of a slight hemorrhage in the upper part of the pons.

Dr. M. G. Schlapp said that the case suggested concussion of the spinal cord, because of the spastic condition of the muscles that had been observed experimentally after injury to the cord.

*Subcortical Tumor; Operation.*—Dr. M. G. Schlapp presented a man who had been successfully operated upon for brain tumor. He had come to the clinic at the Presbyterian Hospital in the middle of July with the fingers paralyzed in a claw position. There was paresis of the face; ankle-clonus and increased knee-jerk were present; there was no disturbance of the tactile sense. The condition began three years ago with a sudden convulsion lasting about half an hour. After about one week the patient developed Jacksonian epilepsy, and the attacks recurred about every three months. There was no history of congenital syphilis. The only history of injury was of being struck on the head by a barrel ten years before. A diagnosis of subcortical tumor was made. Dr. George Woolsey found on operation a cyst, about three inches in diameter, within which and adherent to its wall was a tumor. The latter was peeled out easily, and on examination, proved to be a fibroma. The patient's condition had improved considerably since that time.

Dr. B. Sachs said he had reported some years ago a case of large tumor developing from the wall of a cyst, an occurrence which he did not think was very common. It was probable that the tumor had developed in the wall of an old congenital cyst.

*A Tumor Formation in the Region of the Coccyx.*—Dr. I. Abrahamson presented a child of one month with a tumor in the region of the coccyx, which had been noticed immediately after birth. There was no wasting and the reflexes of the lower extremities were normal. The tumor was situated at the extreme end of the coccyx, and the skin was freely movable over the tumor. There was no cleft in the vertebral column and no pulsation. The tumor appeared, on rectal palpation, to lie between the rectum and coccyx. Only on violent crying did the tension of the tumor vary. It was certainly not a spina bifida.

Dr. Robert Abbé said that it appeared to belong to the presacral tumors—really fetal remains innocent in nature, though sometimes reaching a great size. They were easily removed, but some of these tumors had been known to shrink and almost disappear spontaneously.

*A Specimen of Meningocele.*—Dr. M. G. Schlapp presented the brain of a child who lived three days, and died from some unknown cause. The brain showed a meningocele with almost complete hypoplasia of the cerebellum. There was almost complete absence of the pons.

*Discussion on Spinal Cord Tumors.*—Dr. Joseph Collins opened the discussion with a paper dealing with the symptomatology and operability of these tumors. He had collected 70 cases as a basis for his statements. These tumors, he said, were far more susceptible of surgical treatment than tumors of the brain; nevertheless the majority of spinal tumors proved fatal. There were four reasons for this, *viz.:* (1) The inability to diagnosticate and localize these tumors; (2) the nature and extent of these growths; (3) the great danger to life of the operations for their removal, and (4) the inability of securing the patient's consent to operation at a period when such treatment might prove successful. It must be admitted that the clinical picture presented by such cases was not that given in our books as indicative of spinal cord tumors. A fibroma would give rise to symptoms of as great severity as would a most extensive sarcoma. It was much more important to diagnosticate a tumor in the lower part of the dorsal region than to localize it at a single level—in other words, it was much more important to diagnose a tumor of the spinal cord than the structure from which it springs. If the location of the tumor were determined within from four to six inches he thought it would be near enough for the purposes of the surgeon. The common location of these tumors was in the upper and lower ends of the dorsal region. In the 70 cases that he had gathered from the literature, 35 were from the dorsal region, 15 from the cervical, 13 from the lumbar and sacral, and 7 were of widespread distribution—in other words, in 50 per cent. of the cases the tumor was in the dorsal region. In many cases reported the pain was by no means characteristic, and in some cases it was not even a prominent symptom, as had been formerly supposed. From his personal experience and a review of the literature, priapism was an uncommon symptom.

The speaker then reported a case occurring in a man, first seen in September, 1901. Four months previously he had begun to complain of pain in the abdomen, apparently due to flatulent dyspepsia, and relieved when digestion was improved and the bowels regulated. About this time stiffness of the right leg began. Examination showed a Brown-Séquard paralysis on the left side extending above Poupart's ligament. A diagnosis of spinal cord tumor was made, and an operation urged, but not agreed to until about nine months later. The man was then in a pitiable condition, and was taken into the hospital. The tumor was located by Dr. Abrahamson at the tenth dorsal vertebra, and at the operation, done by Dr. Samuel Lloyd, the growth was found at this point. The patient recovered from the operation, and had since then steadily improved, although the long delay in operating precluded the possibility of a cure. The second case was that of a young girl who became ill with what was supposed to be pneumonia in December, 1898. She remained in the hospital over three months, and the following summer the diagnosis of Pott's disease was made at another hospital. The following autumn, on admission to the City Hospital, she was completely paraplegic, and had enormous bedsores. Subsequently she died in the Montefiore Home, and the autopsy revealed a sarcoma at the level of the seventh dorsal segment. In the 70 cases collected surgical operation had been undertaken in 30, with a successful result in 12, partial success in 8, and unsuccessful in 10 cases. Of the 10 unsuccessful cases death occurred in 4 cases from sepsis and septic meningitis, while in 4 others there were collapse, exhaustion, shock and hemorrhage. In 21 cases the nature of the tumor was stated. Of these 21, 4 were fibromata, 12 were sarcomata, 3 were endotheliomata, and 1 was a myo-lipoma. According to the records 44 of the 70 cases might have been operated upon. Fourteen cases gave autopsy records which indicated that they could not have been operable.

*Report of a Case of Spinal Cord Tumor Operated Upon.*—Dr. Joseph Fraenkel reported this case. The patient was a woman of forty years. Three years and a half before coming under his observation she began to have slight pain in the right hypogastric region, and some time later in the buttock on the same side. Still later a painful area developed in the perineum. After all sorts of treatment she came under his care. At that time the right lower extremity was flexed, and on attempting to put her on her back the movement increased the pain in the areas referred to. There was retention of urine and obstinate constipation. The right knee-jerk was absent, while the left jerk was normal. Both plantar reflexes were absent. There was a curious trophic edema of the right buttock. The diagnosis of spinal cord tumor was made, situated in the cauda equina. The first nerve implicated must have been the iliohypogastric, explaining the first area of pain. The patient was operated upon last April in the

New York Hospital by Dr. Frank Hartley, and she reacted with difficulty. A tumor was found between the strands of the right half of the cauda. It was a reddish, pultaceous mass, which proved on microscopical examination to be a fibrosarcoma. Certain symptoms improved after the operation, but the patient died about two months later.

*Report of a Case of Spinal Cord Tumor Operated Upon.*—Dr. I. Abrahamson reported this case. The patient was a man of sixty in whom the first symptoms had developed in February, 1900. The first symptom was a coldness and numbness in the fourth and fifth toes of the left foot at night. Later, the right foot and leg were similarly affected. On March 15, 1902, he first came under the speaker's observation. On admission to the Montefiore Home, May 12, 1902, there were weakness and atrophy, most marked on the right side. The extremities were flaccid, with some tendency to contracture and a reaction of degeneration. The thighs were flexed, adducted and inwardly rotated. The knee-jerks were lively. There was total loss of voluntary power in both lower extremities. There was a diversity of opinion as to whether the tumor was extramedullary or intramedullary. His own diagnosis was a tumor of the spinal cord at the level of about the sixth dorsal vertebra, and this was confirmed at the operation. Unfortunately, secondary infection occurred, and the patient died as a result of this three weeks after the operation.

Dr. M. Allen Starr said that in his paper, read before this Society in 1895, he had collected from autopsy records 123 cases of spinal cord tumor. Out of this number there were 100 with sufficiently accurate records to allow of the statement that in 75 per cent. of the cases surgical interference should prove successful. Since that time Bruns had published an excellent article, and Schlesinger still more recently had published a paper containing 400 cases of spinal cord tumor. Of this large number there were apparently about 60 per cent. which were operable. Schlesinger's statistics showed that over one-fourth of these tumors were sarcomata, whereas in his own paper most of the tumors seemed to be sarcomata or fibromata. Of Schlesinger's cases 64 were tuberculous, 44 were cysts, 33 were fibromata, 28 were gummata, and 20 were gliomata. The speaker said he had seen 101 brain tumors and 10 spinal cord tumors. Of his 10 cases of spinal cord tumor only 6 had been operated upon. Two of the remainder were gummata with distinct Brown-Séquard symptoms, and subsided gradually under antispecific treatment. The other two had not been operated upon because the diagnosis had not been made early enough and with sufficient accuracy, and the autopsies showed in both that the operation would have been unsuccessful. Of the 6 patients operated upon, all died; 2 of meningitis, 2 of bedsores and 2 of collapse. Dr. Pearce Bailey was, therefore, to be congratulated on the great success attained in the case he had presented to the Society this evening. He would insist upon the absolute necessity of early diagnosis and early operation, a statement which had been fully borne out by the published experience of Dr. McCosh. Although Dr. Collins had called attention to the unreliability of pain as a symptom, it should be noted that in 5 out of the 6 cases reported here this evening, pain had been a rather prominent feature. Moreover in Schlesinger's cases pain was a prominent symptom. He did not see just how the differential diagnosis from meningomyelitis could be made unless pain were present. Schlesinger had also called attention to priapism as one of the common symptoms indicative of spinal cord irritation.

Dr. B. Sachs said that a number of years ago he had expressed himself as being in favor of early interference in these cases of spinal cord tumors, and his further experience had only strengthened this opinion. Reference was made to two of his cases, one operated upon two years, and the other three years ago, both of which were doing well. He would, therefore, insist that operative interference should be urged just as soon as the diagnosis had been made. He agreed with Dr. Collins that too much prominence had been given to the question of localization of spinal cord tumors. An important and significant fact in the history of these cases was that the affection remained unilateral for a very considerable time, and when it became bilateral the symptoms of affection of both sides quickly became apparent. In those cases in which the symptoms were either sensory or motor root symptoms, and in which all of the severer spinal cord symptoms remain in abeyance for a long time, it was probable that the neoplasm was extradural. A rather sudden development of general myelitic symptoms following upon symptoms which had been unilateral for a considerable time, pointed very strongly to spinal cord tumor.

Dr. Walton of Boston said that Dr. Collins' statistics and the discussion emphasized the large proportion of operable spinal tumors and the large percentage of benefit from operation upon such tumors. Such considerations should prevent us from erring on the conservative side when the diagnosis of tumor had been established. It was to be hoped that when we formed the habit of including this possibility in every case of eliminative diagnosis tumors would be more frequently recognized at an early stage, when operation promised the most. That this habit was not established fifteen years ago was illustrated by a case at that time under his care, which was seen by so distinguished authorities as Charcot and Seguin, neither of whom suggested the possibility of tumor, both regarding the case as one of myelitis, and recommending such treatment as the cautery, ergot and strychnine. In 1892 Dr. Putnam, under whose care the patient had come, recognized the lesion as a tumor, and the case had been twice operated upon, once by Dr. Keene and once by Dr. Warren, with beneficial results though not cure. That pain was by no means an essential symptom even when the posterior nerve roots had become involved in new growth, was illustrated by this specimen of intradural carcinoma, which had completely destroyed the posterior nerve roots of one side in the cervical region. The case would shortly be published by Drs. Taylor and Waterman. There was no history of pain during the onset of this lesion. The clinical history closely resembled that of the case reported tonight by Dr. Abrahamson, *i.e.*, atrophic paralysis of the upper, with spastic condition of the lower extremity. Dr. Collins had alluded to the fact that fibromata might cause more pain than tumors of this class, a fact illustrated by the case Dr. Putnam had reported, in which Dr. Warren removed a fibroma lying free within the dura, a longitudinal section of which, from the laboratory of Dr. Taylor, was shown. In this case violent pain, specially including abdominal pain, was the prominent feature. In the microscopical specimens exhibited was included a cysticercus in the substance of the cervical spinal cord in a case of tabes. He had happened upon this while working with Strümpell in 1880. There were no symptoms referable to the cysticercus. If neurologists gave more attention to the possibility of the presence of spinal cord tumors the diagnosis would more often be made at an early stage. He would agree with Dr. Collins that pain was, to say the least, not an essential symptom. In this connection he presented specimens from two cases. One was a case of syringomyelia with pressure on the posterior roots, in which there was no history of pain. The other case was a carcinoma pressing upon and destroying the roots on one side, and also without pain. Dr. Walton also showed a longitudinal section of a fibroma that had been removed by Dr. Warren with great ease. In this last case the pain was excruciating.

Dr. Robert Abbé said that he had seen that very day the patient reported to the Society last year, and upon which he had reported three years ago. The patient was steadily progressing, and there was nothing to indicate any active disease. The tumor was a large one, and at the time of operation involved the cord, so that it was necessary to curette some of the cord away. There had been no increase in any of the cord symptoms. The arms, which were formerly paralyzed, could now be raised to the head. In sarcomata of the spinal cord he thought the growth was slow as compared with sarcomata in other parts of the body. Sometimes a tumor of the spinal cord might grow for a long time, and yet give rise quite suddenly to symptoms. All of his cases of spinal cord tumors had been among males. The pain that he had seen in these patients had been distinctly of a rheumatic rather than of a neuralgic type. He did not quite understand why so many of the reported cases had proved fatal from infection or from shock, because it seemed to him that the operation should be conducted rapidly and with considerable safety. The hemorrhage in these cases was apt to be almost wholly venous, and practically it could be only controlled by pressure. A little strip of gauze pressed into the hollows as the laminæ were opened up would control the hemorrhage, and would generally prevent any great degree of shock. The dura should be opened at once if the tumor were not encountered, and should be split up for any distance necessary, and subsequently sutured with fine catgut. The escape of fluid did not seem to him of special importance; the leakage had always been slight, and had lasted only eight or ten days. The infection of these wounds in the past he thought had been largely due to the use of impure catgut.

Dr. Collins explained that he had not meant to say that pain was not an important diagnostic symptom, but he wished to call attention to the fact that the pain was not by any means characteristic as would appear from the descriptions given in the books.

# CHICAGO NEUROLOGICAL SOCIETY

October 23, 1902.

The President, Dr. Daniel R. Brower, in the chair.

*Myasthenia Gravis.*—Dr. Harold N. Moyer presented a patient, twenty-three years of age, whose early history was negative, except an undetermined infection of exanthematous type. The patient spoke of having had two attacks of measles about three years ago, but was not very ill. The attacks were near the time when his first trouble with the eyes developed. At that time glasses were fitted for diplopia. Dr. Pusey fitted the patient with his first glasses, and he would report the ocular findings.

Dr. Pusey said the patient was first seen by him November 6, 1899, and at that time he complained of double vision. His vision in the left eye was 6-9; in the right eye 6-6; with minus one sphere with half a cylinder his vision was 6-5 in the left eye, and 6-5 in the right eye. At that time he had esophoria of 22 degrees, with right hyperphoria. The esophoria later became exophoria. On the 8th of December, one month later, he worked all day until midnight, with no blurring of vision, no diplopia at the time. His esophoria had disappeared, but he had slight hyperphoria. On Jan. 4, 1900, he had esophoria of one degree, some hyperphoria. July 3d, 1900, Dr. Pusey saw him again, when he complained of double vision. He had exophoria of eleven degrees, with positive divergence of the eyes. August 6th he was refracted again carefully with a mydriatic. The refraction had changed 3-4 D., he having become more myopic with 1-2 D. cylinder at different axes. He then had an exophoria of eleven degrees. He disappeared on the 6th of July, 1902, and was not seen again until the 26th of September, when he complained of double vision all the time, and inability to converge the eyes. Dr. Pusey noticed paralysis of the internal recti muscles. There was no limitation of motion in any direction except the paralysis of convergence, also paralysis of motion on either side on the part of the internal recti muscles. Six weeks ago, there was limitation of motion of the muscles in every direction except in one of the external recti.

Dr. Moyer saw the patient September 8th, at which time there was a marked general weakness, and he complained of stiffness of the lower extremities, feeling as though his knees were bound, as he expressed it. He had some pain for a time, but this gradually disappeared, and was never very intense. On two or three occasions, since September 8th, he has had marked ptosis, more in the left eye than in the right. It would last for two or three hours at a time, or for a couple of days. It has never been in both eyes at the same time. Until the middle of September, there has been progressive loss of strength, which has continued to the present time. There has been no marked increase in weakness in the last two or three weeks. The eye-grounds are normal. Aside from the extreme muscular weakness, there are no objective signs. There is no swaying with the eyes closed. The knee-jerks are not marked, but they are elicited readily. The superficial jerks are all present. The pupils react to light. At the present time there are no disturbances in pain sense; his sense of feeling is normal. It is simply a pure muscular weakness without appreciable muscular atrophy. There is no disturbance in his general health. His digestion is normal; his bowel movements are normal; his appetite is good; he sleeps well, but the muscular weakness is very pronounced. He has numbness in the index finger; the right hand is a little stronger than the left.

The facial muscles are weak. A difficulty of which he complains is a weakness of his jaw muscles; when he eats, his muscles get tired.

Dr. Goodkind asked whether there was any disturbance of speech, to which Dr. Moyer replied that the voice seemed peculiar, but the patient said it was unchanged.

*Cerebral Syphilis, Dementia with Nuclear Degeneration of some Cranial Nerves and Atrophy of One-half of the Tongue.*—Dr. Moyer presented a patient, thirty-seven years of age. Four years ago, August, 1898, he went to Dr. Ingals for paralysis of the vocal cords. At that time he was very hoarse. He learned from Dr. Ingals that there was complete paralysis of one vocal cord, while the other moved. He was given strychnine and local treatment, shortly after which the paralysis disappeared. A year later the paralysis returned. This was two years ago. He was well until the summer of 1901, at which time he did not feel as well as he had previously. There was nothing peculiar in his condition until the autumn of that year, and that time he had what his wife described as " a spell." Sitting at the table he tipped over some glasses, staggered, and could not find his way about. At this time he was working very hard at his occupation of machinist. He improved until November, after which he became very tired and wanted to lie down most of the time. He slept a great deal. He ceased working last November. About that time he began to see double, the diplopia being vertical. In February of this year his right eye turned outwards. At this time the diplopia was lateral. He then developed pain over the right eye, which later shifted to the top of his head. He consulted an advertising eye specialist who "pulled" the eye straight and treated him for one month. Pain then shifted to the back of the head. He resumed his work again during the Spring, but did not remain at it very long. He could not work effectively; he could not keep his mind on what he was doing, and he made mistakes, and finally had to stop work. In June he went to the country, when his throat became worse again. He had some paralysis of the vocal cord, with hoarseness, which disappeared. A few weeks ago his mind began to wander, and it was noted that his memory was impaired. He had difficulty in swallowing, and at night has been wetting the bed. At the time Dr. Moyer first saw him he had a typical Argyll-Robertson pupil on the right side. The light reaction was present in the left eye, but was sluggish. Accommodation reaction was normal in both eyes. There was complete paralysis of the right external rectus. The eye-grounds were normal. The movements of the tongue were defective and tremulous. There is a history, although it is somewhat indefinite, of specific infection dating back to ten or eleven years. Some of the symptoms have disappeared under specific treatment. He has well-marked atrophy of the left side of his tongue. Under liberal doses of the iodides great improvement in the symptoms followed, particularly of the eyes. His memory is better; also his speech.

Dr. Brower asked as to the condition of the reflexes, to which Dr. Moyer replied that they were exaggerated, more so now than when he saw him last.

Dr. Brower asked whether there were any distinct ankle-clonus, to which Dr. Moyer replied there was not.

There were two features of striking interest in this case, the nuclear degeneration, and dementia. The patient has had no emotional exaltation or mental depression. His mind has been weak and feeble; he could not remember where he put things; he could not go to a neighboring store on an errand without forgetting it before he got there. The association of nuclear degeneration with dementia Dr. Moyer had not seen before. The atrophy of the tongue was organic. The reason he presented this case in connection with the other was because of the somewhat striking similarity in the ocular conditions of the two patients, yet their etiology and pathology were very different. There is little doubt as to the specific history in this case, according to the statements of the physician who treated the patient. Dr. Moyer thought the diagnosis to some extent had been cleared up by the marked improvement, both in the physical symptoms and mental state, under specific treatment. At first, when he saw the patient, and noticed the atrophy of the tongue, and paresis of the eye muscles, he thought he had to deal with a case of myasthenia gravis.

Dr. Patrick said he had treated only two cases of myasthenia gravis, and in them strychnine had accomplished more than anything else. He did not think either of his cases was typical. The patient was given one-sixteenth of a grain of strychnine, three times a day, for a year and a half, but had almost ceased taking it now.

Dr. Moyer asked Dr. Patrick whether he regarded his (Moyer's) case as a typical one of myasthenia gravis, to which Dr. Patrick replied in the affirmative and said he thought it would be well for Dr. Moyer to continue to give strychnine month after month.

*Tabes, General Paralysis and Charcot's Joint.*—Dr. S. J. Walker presented a man who came to the Policlinic Oct. 13th. He was forty-four years of age, married fourteen years, and has three living children. The first child his wife had was born dead at term. The other three are living and well, aged respectively eleven, twelve and thirteen. The mother has never had a miscarriage. The patient's occupation was that of a buyer of dry goods. He gives a clear history of specific infection a little over fifteen years ago. Otherwise he gives no history of any serious illness. He complained of no trouble until ten years ago, when he said he had rheumatism in the legs. Upon close questioning him, these pains proved to be of a lancinating character. These pains have continued since, off and on. Seven years ago he had an ulcer at the base of his right big toe, which proved very intractable to treatment, and consequently this toe was amputated. Three years ago he began to notice that he could not walk so well in the dark. For the last three or four years he has had difficulty in urinating, a relative incontinence. He has had no ocular symptoms of any kind, not even transitory diplopia; no ptosis. This was his condition until about seven months ago, when he fell and sprained his right ankle. At this time there was a great deal of pain in the ankle, with swelling. The swelling extended as high as the knee. The pain continued off and on for five or six weeks, and then gradually subsided. Since then he has had no pain whatever in the joint, but the swelling of the ankle joint is still present and in degree about what it was a few weeks after the injury. An X-ray picture was taken by Dr. Hazelton, at the Policlinic, but the man shook so much that it was impossible to obtain a good skiagraph, and the negative was valueless. The enlargement of the foot was not due to bony formation, in the opinion of Dr. Hazelton. He thought it was a cartilaginous enlargement. The patient had been working at his trade up to the time of the injury, and six or seven weeks after the injury, when he returned to work, he noticed impairment of his memory. He had considerable figuring to do on articles of apparel, and was discharged at the end of five or six weeks because of the mistakes he made in his work. Upon examination the Argyll-Robertson pupil was found. He has no knee-jerks. He has analgesia of the legs, and patches of anesthesia upon the trunk. He has slight analgesia extending over the patch of anesthesia on the right side behind. He has the characteristic stumbling speech, and has shown considerable dementia. In short the mental condition is plainly that of general paresis. The patient was exhibited on account of the rather unusual combination of diseases and the Charcot's joint. The diagnosis was made of tabes with perforating ulcer of the foot and Charcot's joint of the right ankle, and finally general paresis supervening on the tabetic affection.

*Lead Palsy.*—Dr. Walker also presented a man who came to the clinic on October 13th. He is thirty-three years of age, and works in a planing mill. He has five children, all of whom are well and healthy. His wife has never had a miscarriage. The family history is fairly good. The patient has never been exposed to metallic poisoning, so far as he knows, nor to arsenic, nor has he been subjected to any drug intoxication. Three years ago he was at the Alexian Brothers' Hospital for three weeks, and was thought to have consumption. A careful examination at the present time does not reveal any lesion in the lungs. He has never had any infectious disease, and has never been seriously sick. He considered himself perfectly well until about fifteen weeks ago, when he noticed swelling in his wrists and in his ankles, with some pain. The pain passed out of the ankles almost immediately, and after two days in bed he was able to walk around. About the second day he noticed weakness in his wrists and hands, and at the end of three or four days he suddenly lost power in his wrists. In other words, he had double wrist-drop. Since then his condition has been about the same; no paresthesia; no subjective symptoms of any kind except wrist-drop. Upon examination Dr. Walker found that he had double wrist-drop with the supinator involved on the right, but not on the left side, and paralysis of the extensors more marked upon the right than the left side. Patient is right handed. There is partial reaction of degeneration in all the extensor muscles, and supinator on the right side, this being more marked on that side than on the left. There is no objective sensory disturbance. His knee-jerks are equal and normal. The one pupil responds to light and to accommodation (the other eye is artificial). He looks cachectic, and an examination of the blood shows 75 per cent. hemoglobin, reds 3,568,600, whites 2,100.

Dr. Walker spoke of one thing which may be a very valuable indicator in cases of lead poisoning, and that is the presence in the red cells in nearly every case of lead poisoning, if the case is at all severe, of little granulations. These are found in every field in any severe case of lead poisoning. From a medico-legal standpoint, Grawitz, of Berlin, who does much work on blood, regards these small granulations of great value, because when once present they do not disappear until the lead is out of the system. Dr. Walker examined this patient's blood for these granulations in the red cells, and was unable to find them. He examined five or six specimens, each one of which was negative. This man is not addicted to excessive use of either alcohol or tobacco. At most, he drinks four or five glasses of beer daily, and usually not that much. He was closely questioned as to exposure to metallic poisoning, and Dr. Walker had not been able to elicit any cause for the neuritis. It corresponded in distribution to lead palsy, except that the supinator on the right side was involved. The patient has a questionable lead line. The condition of the gums is so bad, almost scorbutic, that it is difficult to say positively that there is a lead line. He has never had lead colic, although that is usually the rule preceding lead palsy.

Dr. Brower said that some people were sensitive to lead poisoning at certain times. He mentioned how it was possible for one to have lead poisoning in Chicago. In this city, in certain parts, the water pressure at certain hours of the day was so low that the water did not reach the upper parts of houses; the pipes are empty of water for hours at a time, then the water is turned on, the pipes are coated with the oxide of lead, which is washed off, and people were poisoned with lead in this way. He asked about the water supply of the factory in which the patient worked, to which Dr. Patrick replied that the man does not come in contact with paint, bisulphide of carbon, or gas.

Dr. Moyer suggested the possibility of poisoning by food.

Dr. Patrick said the patient has never had lead colic, and lead palsy, without preceding lead colic, in cases of chronic poisoning, is unusual.

Asked in regard to the treatment, Dr. Walker stated that it consists of hot baths, the use of iodide of potassium, and massage.

Dr. Brower said a middle-aged man, who had wrist-drop, came under his observation a few years ago. He could not ascertain the source of the lead poisoning until the patient had been under treatment for some time, then he found that he was having his hair dyed.

Dr. Goodkind asked whether the patient had any optic atrophy.

Dr. Walker replied that he had not.

# Periscope.

## NEUROLOGISCHES CENTRALBLATT

(Vol. 21, 1902, No. 17. September 1.)

1. Early Symptoms of Tabes and the Achilles Reflex. S. GOLDFLAM.
2. Syphilitic Epilepsy. J. A. FEINBERG.
3. Dietetic Treatment of Epilepsy. H. SCHNITZER.
4. Subcortical Origin of Isolated Muscle Cramps. J. SORGO.

1. *Tabes and Achilles Reflex.*—The value of the loss of the Achilles reflex as symptom of initial tabes is confirmed by the investigation here set forth. The earliest symptom of tabes and the most constant, is the presence of the lightning-like pains.

2. *Syphilitic Epilepsy.*—A report of six cases of epilepsy, classified as epilepsia luetica. These cases are divided into two general types: those without other cerebral phenomena, and those in which neural manifestations either precede or follow the epileptic attack. Two cases of the first type are given without autopsy. Three of the cases of the second type presented the Jacksonian type of epilepsy. Another case of the second type developing after a trauma, at autopsy there was found an abscess of the left frontal area.

3. *Dietetic Treatment of Epilepsy.*—A study of the effect of Balint's modification of the treatment of epilepsy by withholding salt suggested by Toulouse and Richet is carefully made. The administration of the bromides in the bread as a substitute for salt and the other salt-free diet is strictly adhered to. The results, while positive, were not so favorable as those reported by Balint. In two cases the patients were entirely free during the experiment, in ten cases a very marked, in two cases a not so decided improvement, and in two cases no improvement at all. The bread, at first very acceptable, was later refused by the patients. The author suggests that in chronic epilepsies, a six to eight weeks' course of treatment from time to time might be carried out with hopeful results.

(Vol. 21, 1902, No. 18. September 16.)

1. A New Nucleus in the Formatio Reticularis of the Upper Quadrigemina. W. v. BECHTEREW.
2. The Lumbo-Femoral Reflex. W. v. BECHTEREW.
3. Myotonic Pupillary Movements. A. SAENGER.
4. Infraspinatus Reflex; A Heretofore Undescribed Reflex. STEINER.
5. Further on the Supra-orbital Reflex. D. J. MCCARTHY.
6. Anxiety in Hysteria and Neurasthenia. AUG. DIEHL.

1. *A Distinct Nucleus in the Formatio.*—A distinct grouping of ganglion cells in the upper parietal region immediately behind the posterior quadrigeminal bodies, is given the name of *neucleus centralis superior* to distinguish it from the other cell groups in the same region.

2. *Lumbo-Femoral Reflex.*—In patients with pathological lesions of the dorsal cord associated with heightened reflex activity in the lower extremities, a tap of the percussion hammer over the upper sacral or lower lumbar region with the patient in a half-flexed attitude, results in a simultaneous reflex activity of the thigh muscles and also the erector spinæ. Abduction of the thigh alsooccurs in some cases.

3. *Myotonic Pupillary Movements.*—Saenger reports a woman of thirty-five years suffering from headaches, and who exhibited a marked dilatation of the pupil. After contraction of the pupil by accommodation, convergence or by energetic contraction of the orbicularis palpebrarum, it took as long as ten minutes before the pupil regained its usual dilated condition. Contraction of the pupil was likewise slow. He regards the condition as of local character, *i.e.*, in the iris itself and analogous to the myotonic contraction of Thomsen's disease.

4. *Infraspinatus Reflex.*—A new reflex elicited by percussion of the infraspinatus area and resulting in an extensor movement of the arm, with lateral rotation. (This reflex was described in an article on the "von Bechterew's Reflex," in the JOURNAL OF NERVOUS AND MENTAL DISEASE, May, 1901, by Dr. W. Pickett, Reviewer.)

5. *Supra-orbital Reflex.*—A controversial paper between McCarthy, von Bechterew and Hudovernig as to the nature of the supraorbital reflex.

6. To be continued. McCARTHY (Philadelphia).

### REVIEW OF NEUROLOGY AND PSYCHIATRY

(Vol. 1, 1903, No. 1. January.)

1. Local Panatrophy. SIR WILLIAM R. GOWERS.
2. Psychiatry in General Hospitals. SIR JOHN SIBBALD.
3. The Relative Frequency of Disseminated Sclerosis in this Country (Scotland and the North of England) and in America. BYROM BRAMWELL.

1. *Local Panatrophy.* Sir William reports on a case seen by him in 1885, resembling a variety of spinal muscular atrophy, yet different. He suggests the term *local panatrophy.* In certain areas of the trunk, limbs, or face, which vary in diameter from that of a nut to that of an orange, or larger, there seems to be a wasting of all the subcutaneous tissues down to the bones, with slight change also in the skin, which is there distinctly thinner and slightly discolored. The aspect of the areas may be described as that of a subcutaneous excavation. They seem to be distributed quite irregularly, without apparent relation to the muscles or to the nerve distribution. Where the process is considerable the muscular tissue shares the wasting, but seems not entirely to disappear, and the electric irritability of that which remains is normal. In a case of Dr. Harry Campbell's recently shown at the Clinical Society there were similar patches in the right foot in which the bones seemed also to have atrophied.

2. *Psychiatry in General Hospitals.*—This is an interesting general article on the relationship of psychiatry to neurology and the needs for a closer association of the two studies. The causes that have led to the separation are considered and the reasons why patients with mental diseases should be provided for in general hospitals fully discussed. He quotes Maudsley as saying that "mental disorders are neither more nor less than nervous diseases in which the mental symptoms predominate, and their entire separation from other nervous diseases has been a sad hindrance to progress." A number of interesting figures are given showing the kind of psychiatrical instruction given in Germany where under the impulse of Griesinger great advances had been made in the treatment of mental cases in hospital wards connected with the universities.

3. *Disseminated Sclerosis in Scotland and in America.*—Dr. Byrom Bramwell speaks of the relatively greater incidence of disseminated sclerosis in Scotland and the north of England than in America. In his statistics of hospital and private cases, the figures show one in 58 nervous cases to be disseminated sclerosis or contrasting his hospital experiences with his private work, the proportions are one in 54 and one in 62. He draws a comparison with the statistics as given in the JOURNAL OF NERVOUS AND

MENTAL DISEASE (vol. 29, May, 1902, p. 288), where in a discussion before the New York Neurological Society it was shown that only one in 221 nervous cases was of multiple sclerosis. Thus in Scotland and the north of England this disease appears to be three and one-half times as frequent as in America. JELLIFFE.

### LE NEVRAXE

(Vol. 4, 1902, Fascicle 1.)

1. Researches on the Central Sensory Tracts. II. The Central Tract of the Nuclei of the Posterior Columns, or the Central Medullo-thalamic Tract. A. VAN GEHUCHTEN.
2. The Endocellular Reticulum of Golgi in the Cerebral Cortex. S. SOUKHANOFF.
3. The Posterior Root of the Eighth Cervical and First Dorsal Nerve. A. VAN GEHUCHTEN.
4. The Protoplasmic Prolongations of the Nerve Cells of the Spinal Cord of Higher Vertebrates. S. SOUKHANOFF and F. CZARNIECKI.
5. Lesion of Cauda Equina. Contribution to Study of Centers of Micturition, Defecation, Erection, Ejaculation and of the Anal Center. A. VAN GEHUCHTEN.

1. *The Central Tracts of the Posterior Column Nuclei.*—Van Gehuchten contributes an able and lengthy article on the subject of what becomes of the fibers having their origin in the nuclei of the columns of Goll and Burdach. He first brings out in a clear manner the divergent views of Forel, v. Monakow, Bechterew, and others with reference to the origin and destination of the fibers that make up the lemniscus, both median and lateral. His own researches were on rabbits in whom the nuclei cuneatus and gracilis were destroyed by puncture and scission and he shows among other things: (1) That there are no uninterrupted crossed fibers that go from the nuclei of the posterior columns to the cerebral cortex (in opposition to Tschermak); (2) the fibers that have their origin in the nuclei of the posterior columns terminate for the most part at least, in the lateral nucleus of the optic thalamus of the opposite side, thus constituting an *ascending medullary thalamic* tract; (3) this ascending medullary thalamic tract is exclusively crossed; (4) the fibers of this central tract that originate in the nucleus gracilis appear before those coming from the nucleus cuneatus. The former make up the greater part of the preolivary nucleus; the others the median part, all of the interolivary nuclei and the intra and periolivary fibers. In the median lemniscus it is difficult to separate the fibers from the nucleus gracilis and the nucleus cuneatus. The former seem to preponderate in the external portions of this part of the lemniscus, the others in the internal part; (5) the method of Marchi does not permit of the precise determination of all of the connections of the ascending fibers with the nuclei of the bulb, the pons, or the mesencephalon. All that can be asserted is that during its course the medullo-thalamic tract sends a portion of its fibers to the corpora quadrigemina. Whether they go to the inferior corpus, as claimed by Bruce, Wallenberg and Rothmann, or to the superior corpus, according to Ferrier and Turner, he does not determine.

2. *The Endocellular Reticulum in the Cortex.*—Rabbit's brain is prepared in Veratti's fluid; see original for technic, which is complicated. The network is found to be perinuclear. It does not reach the periphery of the cell. Its general arrangement is that of the cell outline, and the author believes it to be interpreted on some such structural basis as that described by Holmgren and Retzius as "intercellular canals or pores." Butschli's "foam structure" may account for some of the picture.

3. *Posterior Root of the Eighth Cervical and the First Dorsal Nerve.*—

This is another contribution by Van Gehuchten to the study of the central termination in the nuclei of the posterior columns of the peripheral sensory nerves. The study of the degenerated tracts up to the nuclei show that they occupy a position slightly anterior and just external to the column of Goll.

4. *Prolongutions of Nerve Cells in Cord of Higher Vertebrates.*—The Golgi methods show in adult rabbit cords three types of cells: (1) Bipolar cells with two dendrites at one pole and one from the opposite pole. Irregular varicosities are typical of both dendrites. Gemmules are also present at long intervals and are few in number. (2) Fusiform cells with many dendrites which are literally covered by gemmules. (3) Cells with four dendrites, occupying the white matter. These are extremely irregular in general contour.

In the anterior horns the dendrites fall into three general classes. (1) Some are of uniform caliber; (2) others, more numerous, have moniliform swellings, and (3) moniliform dendrites. The nerve cells of the cord differ markedly in the matter of prolongations from those in the cortex. They are more often varicosely swollen. The gemmules are fewer and vary more widely in shape and size. The authors describe in the cord a unique type of offshoots intermediate between the gemmules and protoplasmic processes.

5. *Lesion of the Cauda Equina.*—(Continued article.) JELLIFFE.

### IL MANICOMIO

(Vol. 18, 1902, No. 1.)

1. Some Notes on Pathological Anatomy in the Insane.—G. ANGIOLELLA.
2. The Basic Unity of Psychopathic Processes.—F. DEL GRECO.
3. Oscillations of the Metabolic Processes in the Epileptic.—N. ALESSI AND A. PIERRI.

1. *The Pathological Anatomy of the Insane.*—The most prevalent lesion of the brain, corresponding to the frequency of general paralysis, is chronic fibrous leptomeningitis, associated with periencephalitis, the latter being characteristic of general paralysis of the insane. In 450 autopsies, this lesion was found in 108, namely in 94 men and 16 women. Among the various other lesions associated with the leptomeningitis, the author found meningeal hemorrhage, edema of the brain, softening of certain portions of same, also endarteritis deformans, insufficiency of the aorta, a large proportion of chronic nephritis, and most of all, omitting many other affections, lobular bronchopneumonia, namely, 46 cases. The author calls special attention not only to the multiplicity of the associated lesions, but also to the variety of their combinations, the former circumstance going to prove the author's contention that paralysis of the insane is a general disease, affecting not only the nervous system, but the whole organism through the vascular apparatus, which latter, surcharged with "toxic" blood, undergoes changes such as chronic inflammation; this in its turn causing alterations in various organs, and parenchymas of same; these therefore suffer in their nutrition, degenerate, become inflamed or undergo necrosis. As regards the variety of lesions it may possibly be explained by a particular susceptibility of certain organs in some individuals to alterations produced in the blood of the patients.

It is worthy of note that only in one case were there encountered syphilitic lesions, namely gummatous cicatrices of the liver; it must, however, be stated that in general it is rather difficult to discover at the autopsies of paralytics clear and indubitable traces of syphilis. For those cases in which the etiological factor of syphilis, alcohol or other poisons can be excluded, the author finds the causation of the affection in overwork, exhaustion (what the Frenchmen very appropriately designate as *surménage*), which may be supposed to bring about the appearance of abnormal chemical products or an insufficient elimination of the normal metabolic processes, thus producing intoxication of the blood with the resulting vascular and parenchymatous lesions. It is claimed by many investigators that syphilis and alcohol are not sufficient by themselves to cause general paralysis, while the association of overwork or nervous exhaustion with these poisons frequently results in bringing about this condition.

Next in frequency the author finds hyperemia of the meninges and of the brain substance, largely of the cortex, as encountered in 66 cases; 52 men and 14 women. In six of these cases the hyperemia was the essential morbid condition producing clinically the picture of acute delirium. Another group of cases showed chronic or transitory mania. In these there was also found hyperemia in other viscera, in the kidneys, lungs, gastro-intestinal tract, spleen, *etc.*, showing that the intoxication was general. Still another category included cases in which the cerebral hyperemia existed only as a local manifestation of an infectious disease in other organs; others again were epileptics who died during an epileptic attack; some suffered from cardiac affections, general debility, *etc.* Cerebral anemia was met with in but a few cases, in connection with general anemia and chronic exhausting affections, preëminently tuberculosis of the lungs and other organs.

Senile atrophy of the brain was seen in 11 cases, 5 men and 6 women, in connection with diffuse atheromatosis, external hydrocephalus, pulmonary and cerebral edema, cardiac hypertrophy, *etc.* It is, however, difficult to state positively where physiological senile involution ends and where the pathological process begins, although clinically the difference does exist, and deviations from physiological senile involution can be detected in life and classified similarly to what may be observed in the insanities of puberty and menopause.

The author calls special attention to a very interesting fact, namely, the comparative rarity of focal affections of the brain in mental diseases; thus he records only six cases of cerebral hemorrhage (conected, however, with acute leptomeningitis, bronchopneumonia, *etc.*), and 6 cases of softening of the brain; in a simple epileptic, in a deaf-mute epileptic idiot, in a seventy-two year old woman with senile melancholia, softening of the corpus striatum and internal capsule in dementia, in an indefinite case of epilepsy (?) and in one of senile hypochondriasis. Then there were 2 cases with abscess of the brain, 3 cases of glioma, one case of ecchinococcus (10 cysts of various sizes), accompanied by diffuse acute leptomeningitis; also one case of syphilitic gumma (Jacksonian epilepsy), and one sarcoma of the meninges. The author is inclined to attach comparatively little importance to focal lesions: hemorrhages, neoplasms, softening, suppuration, in the production of mental affections. Of the accidental diseases he found acute catarrhal pneumonia (102 cases), pulmonary tuberculosis (71 cases), croupous pneumonia (42 cases), pleurisy (29 cases), endocarditis (40 cases), hepatitis, pericarditis, chronic intestinal catarrh, peritonitis, chronic nephritis (75 cases), *etc.* Some of these affections are not to be considered as simply accidental in the course of the mental malady, others may be so considered; to the first group belong first of all affections of the kidneys (75 nephrites in 450 autopsies), then aortitis and endarteritis, and others. The second group of cases simply shows how readily the poor insane with his abnormal or debilitated nervous system becomes peculiarly vulnerable and falls an easy prey to various infectious agencies, the most pernicious of which is tuberculosis, in all its forms; then the various pulmonites, pleurisy and pulmonary gangrene. On the other hand it is well known with what rapidity epidemic diseases spread in asylums.

As regards the relation between infection and insanity, it is worthy of note that some insane present a truly remarkable resistance to certain chronic intoxications, and especially to tuberculosis. There are observed cases in which the tuberculous process remains stationary for years and years, and the patient, after a prolonged confinement in bed, debilitated, exhausted, emaciated and profoundly anemic, dies eventually of slow marasmus. This is one of the facts we are unable to explain in the insane.

2. *The Basic Unity of Psychopathic Processes.*—A lengthy psychological sketch on insanity and degeneration which does not lend itself to abstraction.

3. *Oscillations in the Metabolic Processes of the Epileptic.*—It has long been observed that the epileptics present very frequently facial asymmetry (Laseque), strabismus and inequality of the pupils (Bianchi), also asymmetry in the coloring of the iris (Féré), as well as astigmatism. Tonnius finds left-handedness more frequently among epileptics than among insane. Zucarrelli noted asymmetry in the sides of the thorax. Other abnormalities relate to the length of the fingers, lack of visual acuity, impaired hearing, inequality in the tendon-reflexes, *etc.* This asymmetry in the epileptic is also manifested in his motor and psychic activity; and even though his mentality is frequently precocious, it is usually one-sided, and deficient ethically. Féré defines the character of the epileptic as "*mobil et explosif.*" The author of the present investigation, desirous of verifying whether this morphological and functional disharmony finds a corresponding expression in the metabolic activity of the epileptic, examined for some time the urines of epileptics every day. The subjects were 7 epileptics who presented special variations in their deportment; the examinations extended to between 27 to 37 days. Their food was the ordinary hospital regimen. The patients were free from any visceral affection, and they were given no drugs. The principal examination was directed to urea and especially to phosphoric acid, which is considered as a true index of the activity of the central nervous system. The general considerations drawn are as follows: The quantity of urine at all times was rather scarce, presenting in some cases notable oscillations, especially in four patients who were typical epileptic subjects, irascible, morose, frequently menacing and impulsive. Nor is there noted any special diminution in the quantity of the urine on the days when the patients had epileptic attacks. The specific gravity, always rather high, was generally in inverse proportion to the amount of the urine. Here were observed frequent oscillations corresponding with the curve of phosphoric acid. The reaction of the urine was almost always acid. Neither peptone nor sugar was ever found in any of the urines, and hardly a trace of albumin, in one case. The amount of urea was generally low; whatever oscillations were observed were always in correspondence with the phosphoric acid curve. As regards this latter, the author states that the amount of it was usually rather small, and only sometimes increased to some extent. The variations in the amount of phosphoric acid were very considerable and almost always in correspondence with the excretion of urea. Although not positively so it may be stated in a general way that there was a certain constant relation between the variations in the amount of phosphoric acid, and the epileptic seizures. A close examination of the subjoined tables of minute observations of the cases brings out the fact that rapid and very considerable oscillations are found not only at the intervals, but also on the days when no attacks occur. This the author thinks is due to the fact that alongside with the above mentioned somatic and functional abnormalities there is in the epileptic a certain daily instability of metabolic activity as evidenced by the curves in the various constituents of the urine. This has been seen even in those patients in whom the attacks occur at long intervals. The author contends that the lack of equilibrium in the organo-

genetic forces of the epileptic as manifested by the various asymmetries and by the great variability of the organic and psychic functions stands in direct relation to a certain oscillating condition of the chemical productivity of the various tissues of the body, as especially manifested by the connection between the amount and specific gravity of the urine, and the excretion of urea and phosphoric acid. The erudite author devotes considerable space to the discussion of the present state of the phosphoric acid problem in its relation especially to the activity of the central nervous system.

(Vol. 18, 1902, No. 2.)

1. The So-Called Polyneuritic Psychosis.—G. Esposito.
2. Imbecility and Asexualism.—G. Angiolella.
3. The Suicidal Tendency and Suicide in the Insane.—R. Gucci.

1. *Polyneuritic Psychosis.*—It was Korsakoff of Moscow who first called attention to this psychosis, some time in 1887, since which time the disease has been known under his name. He was the first to prove its existence by the aid of 14 classical observations in which alcohol was absolutely excluded as an etiological factor of the polyneuritis, and instead were found sepsis (puerperal), typhoid, tuberculosis, *etc.* The condition of polyneuritic psychosis is characterized by symptoms of multiple neuritis associated with psychic disturbances such as amnesia, alteration of ideation, with the predominance in some cases of symptoms of irritability and excitement, consciousness being either entirely preserved or somewhat obtunded. All the symptoms may be of variable intensity. The patient retains no hold in his memory over recent events, while he may remember well past occurrences. Outside of the psychic state we find gradual emaciation, loss of strength, weakening of cardiac activity, certain changes in the urine, *etc.* However, it does not seem that all the neurologists coincide as yet with Korsakoff in accepting this clinical picture as a pathological, well-defined entity, and Jolly proposes to designate it as Korsakoff's syndrome. As this psychosis presents a certain similarity with confusional insanity, some authors are inclined to consider it as but one variety of this last, and indeed the points of contrast between these two affections are many—in the etiology, symptomatology, and pathological anatomy. The author presents two cases, one of an alcoholic, the other with a more or less definite history of syphilis and malaria, and rather moderate alcoholism. Both cases presented distinct symptom-complexes of acute confusional insanity. The author devotes considerable space to the psychological and anatomo-pathological considerations of amnesia, especially as met with in the so-called polyneuritic psychosis, and some consideration is given to the obscure and controversial problem of memory. One important fact may be established without any doubt, namely: that the disturbance of memory is a symptom common to all the pathological processes due to toxemia of the central nervous system, and that the variations in this disturbance stand, in some cases, in direct relation to the particular pathogenic agent, as well as to the special individual idiosyncrasy. The amnesia of the polyneuritic psychosis lacks any pathogenic character, and therefore the psychosis as a consequence bears no peculiar individual imprint of its own. In fact no diagnosis of any malady should be based on one or two symptoms, especially when neither the one nor the other is in any way pathognomonic. It is well to remember Nissl's advice as to arriving at a diagnosis of a mental affection: the diagnosis is to be drawn not only from the syndrome of symptoms as they appear but from a full and thorough examination of all the elements which go to make up the characteristic features of the disease, and from the antecedent history of the individual. The author thinks that the disease under discussion is but a part of the

great picture of confusional insanity, as had been asserted by Kræpelin, and cannot be considered as an affection *sui generis* among the infectious insanities.

2. *Imbecility and Asexualism.*—This is a clinical sketch based on the history of a youth, eighteen years of age, of a markedly tainted origin (mother "weak-minded," father stammerer, and derelict in his paternal duties to his offspring). The patient when grown old enough showed neither willingness nor capacity for work; was always weak-minded, and confined on this account several times during variable periods in the asylum. The physical measurements, especially of the head, showed certain abnormalities; he also has slight gynecomastia, flat larynx, fat abdomen, protruding buttocks, and a marked dorso-lumbar curvature. There is a complete absence of testicles, the scrotum hardly visible as a slight tumefaction, soft and delicate, of the skin; instead of the penis, a simple cutaneous appendix with a canal for the urethra, without any distinct features of body or glands, and no trace of any corpus cavernosum. Psychically the patient presents undoubted infantilism, and is much inferior in his mentality to his age, his behavior being that of a boy of a few years old. He demonstrates no affection either for any member of his family or his comrades in the institution. He lacks completely all the sexual instincts and sentiments, in fact he shows a certain degree of aversion toward women, although he seems to understand the difference between himself and the normal male. The question that interests one the most is whether the physical condition of the patient is simply coincident with his mental inferiority, or whether there is a certain connection between the two. It is a well known anatomical fact that atrophy of a certain region of the nervous system, and especially in the cortical zone, is not without influence on the development of the rest of the organism. Now, if a certain portion of the cortex is lacking, all the fibers which originate or gather there will either be absent or atrophy, thus inducing certain alterations in the various regions with which these fibers connect. May the absence or at least atrophy, in the case under discussion, of certain centers and of nervous paths leading to the sexual organs and functions be considered as the cause of deficient mental development? Can we establish a relation between the genital centers, wherever they be located, and the process of mental development, similar say to that between the center of speech and the higher special centers? The following facts seem to answer these questions more or less completely. The rapid increase in potentiality, in intellectual energy accompanied by marked changes in the character is accompanied in both sexes by the establishment of secretion in the genital organs, by secondary sexual characteristics, the appearance of hairy growth over the pubic region, the increase in size of the larynx in man, and the mammary glands in the woman, etc. It is at this age that the imagintion faintly awakens, and the sexual function which is the basis of the most elementary social organism, the family, causes the appearance in the individual of the sentiments of paternity, filial love, *etc.* In the same manner must we expect to find a decadence of the mental faculties, and a change of character in case the sexual functions be extinguished; it is the involution, as it were, of the whole organism which gives rise to such psychic deficiency.

3. *Suicide Among the Insane.*—The problem of suicide in general in its sociological aspect, and of suicidal tendencies among the insane in particular is one which has attracted the attention of alienists for a long time past. Thus Esquirol, Pinel, Marcé, Dagonet, Bucknell and Tuke, Maudsley, Kræpelin and others have all occupied themselves with this most important question, each of them offering his views on the solution of this problem. Latterly the Italian investigators, as Morselli (in his work

"Il Suicidio"), L'Antonini and others, contributed some very valuable information on the subject. The author's study is based on the observation of 132 inmates of an asylum who have shown positive tendencies toward suicide (out of a total of 405). The greatest number, namely, 29, is found among melancholics; then follow those affected with dementia præcox, epileptic insanity, mania, paranoia, and so on down the list. Among those with precocious insanity, the tendency has been observed only during the periods of depression. But of course, it is the real melancholics that present the most classical form of an obstinate tendency to suicide; in them it is continuous, invincible, not to be easily overcome. In them it may even be observed in status raptus. As regards the epileptics there is a greater danger in their impulsiveness as regards the destruction of those around them; but the period following the epileptic "explosion" is marked by such a degree of depression that the patient falls a ready prey to the suicidal tendency; to this must be added the various hallucinations that follow after the attack and that may bring about an irresistible impulse toward suicide, which they frequently commit in the most unexpected and unlooked-for manner. The alcoholic is subject to suicide during grave mental depression associated with various hallucinations. The statistics gathered by the author, covering a period of some sixty years, show that there were but 22 suicides in the asylum (at Florence), a really small number if we take into account that the yearly number of suicides in Italy alone is more than 2,000, and that the asylums shelter insane with irresistible suicidal tendencies; in fact hardly one per cent. of those admitted destroy, or rather succeed in destroying themselves. The author brings forward certain statistics as regards the sexes of these suicides, their ages, their means for the completion of the attempt, the season of the year, and the places where these successful attempts were accomplished—statistics which, we must confess, are hardly of much scientific value, as they are based upon a comparatively meager number of subjects and thus admit of no generalization, the chief value of statistical investigations as regards practical application. Still it must be recognized as a sad fact that suicides do occur in the insane asylums among those patients who are addicted to suicidal tendencies, and such inmates require special care and rigid surveillance on the part of the attendants to frustrate their attempts at self-destruction. ALEX. ROVINSKY (New York).

## MISCELLANY

THE CRIMINAL EQUIVALENT OF INSANITY. Wm. B. Noyes. Medical News, October 11, 1902.

This paper, discussing the obscure problem of so-called moral insanity, presents two cases. The first is the common type of "bad" or "incorrigible" boy, unfortunately a son of wealthy parents, in whose case the association of some mental backwardness and fits of ungovernable temper with numerous moral defects, indicates the presence of a psychosis presenting chiefly moral symptoms. The second case indicates the possible substitution of immoral or criminal tendencies for the common symptoms associated with a mental breakdown. In this case a short attack of acute insanity changed a boy of fairly decent tendencies into a professional crook. The indefiniteness of the legal attitude toward such a case is shown by the fact that he was first sent to Matteawan Asylum for insane prisoners, and later for the same offense to state's prison. A careful analysis of the history of law relating to such cases of partial insanity, shows decided variation of methods and by different states, especially marked in their use of such expressions as "impulsive insanity" and criminal responsibility. A psychological treatment of the subject attempts to analyze defects of the will and reasoning powers. The importance of fixed ideas or obsessions on obscure cases of crimes cannot be too frequently brought to the attention of jurists. The presence of minor eccentricities is important, for there is no hard and sharp line to be drawn between the eccentric and criminal action. Such mental eccentricities are often as clearly stigmata of degeneration as the more definitely understood physical stigmata. The cases of moral defectives may be grouped in three classes: (1) The moral idiot who has not a sign of a moral concept, abstract or concrete; (2) the moral imbecile who knows the concrete but not the abstract; and (3) the "débile" who knows both concrete and abstract, but lacks the "tone of ethical feeling." Such a man knows right and wrong, but will not on that account be kept from committing wrong. WM. B. NOYES.

THE PREVENTION OF DEFORMITY. Wisner H. Townsend, M.D., Journal American Med. Assoc., Sept. 13, 1902.

Practically all deformities due to poliomyelitis or infantile paralysis can be cured. The original lesion does not primarily produce a deformity, but simply a loss of power in the muscles. The contractures come on very slowly. Non-congenital club-foot may be divided into (1) simple acquired; (2) paralytic; (3) traumatic. (1) Simple acquired club-foot includes all cases due to rickets, rheumatism, chorea, hysteria and various other surgical and medical conditions. (2) The paralytic includes all cases due to poliomyelitis anterior, hemiplegia, meningitis, myelitis, progressive muscular atrophy, spastic cerebral and spinal paralysis, compression myelitis, irritation of pyramidal tracts and syringomyelia. Half the deformities are preventable by proper appliances.

W. B. NOYES.

SCHOOL LIFE AND INSANITY. J. S. Lankford, M. D. Medical News, Sept. 27, 1902.

The writer discusses high pressure in the school life of children. Too much is attempted in the school curriculum. If pupils are examined, they will be found excitable, emotional, wakeful, discontented and suffering often from headaches and nervous dyspepsia, the girls showing a decided tendency to hysteria, with here and there spots of anesthesia and hyperesthesia discoverable, and various nervous symptoms connected with their menstruation. Excessive piano practicing produces many victims.

A large number of children come out of school with unbalanced nervous and mental systems, weakened will power and an incurable "hurry habit," engendered by school life. If troubles connected with business or love then develop the boy or girl is ready for serious mental disease. The writer advocates (1) reducing the course of study; (2) developing the body co-equally with the mind; (3) instituting a better classification of pupils and their individual tendencies; (4) object lessons rather than books; (5) introducing industrial training as rapidly as possible. WM. B. NOYES.

HOSPITALS FOR THE NEUROPATHIC AND PSYCHOPATHIC. Richard Dewey. Journ. of the Amer. Med. Assoc., Sept. 27, 1902.

Twenty years ago not a single institution existed in this country for the care of the criminal insane, or for the separate treatment of the epileptic, criminal or alcoholic classes. Since then there has grown up a strong interest in and demand for special and separate care for the acute and curable insane. Criminal asylums, epileptic colonies and hospitals are rapidly springing up. Convalescent homes for nervous and mental maladies are being developed or suggested. Such homes are specially adapted to the treatment of hysteria, neurasthenia, hypochondria, mild melancholia, and harmless paranoia. There is also a large class of organic diseases affecting the brain or spine, with or without some mental symptoms which are not suited to a general hospital, which can be treated in such homes. A psychopathic hospital is needed in every large city for cases of the type of acute or subacute psychoses, but should be located in the quiet suburb. There should be not more than twenty-five patients to each physician, and five patients to each nurse; twelve to thirty patients should be the limit of any one building, and the cases should be carefully classified, secluding the noisy and turbulent.

W. B. NOYES.

UREMIC APHASIA. David Riesman. Jour. Amer. Med. Assoc., Oct. 11, 1902.

The poison of uremia, presumably a form of auto-intoxication, partly associated with disease of the kidneys, acts chiefly on the nervous system, although no organ or tissue escapes. Like hysteria, uremia often presents distinctly focal symptoms, such as hemiplegia, monospasm and aphasia. The existence of uremia does not exclude the possibility of hemorrhage or embolism, since patients with Bright's disease have for the most part friable arteries. Uremic aphasia may be associated with right sided paralysis, or occur alone. Of twenty-nine cases of uremic aphasia in literature fifteen were pure aphasias; fourteen had some co-existing palsy. Children are especially liable to it. Its onset is sudden. The type of attack is generally motor aphasia of the subcortical type. Writing may be affected also.

The degree of aphasia varies from total loss of speech to a slight paraphasia, with or without paragraphia. Of the twenty-nine cases, hemiplegia was present in ten instances; in two facial paralysis and in two right brachial monoplegia. Convulsions may precede or follow the development of aphasia which may be unilateral or general. Hemianopsia may be observed. More frequently amblyopia. The uremic paralysis and aphasia may be so fleeting as to be almost overlooked. It may last five minutes, or twenty-four or forty-eight hours, or sometimes a week. The only lesion demonstrable at autopsy is edema and congestion. The kidney is apt to be the contracted kidney. The cause of the uremia is still unknown, whether a retention of excrementitious substances in the system, or an albumin intoxication, with some structural alteration of the vessels in the area affected. .W. B. NOYES.

SUSPENSION AND CORSETS IN CHRONIC MENINGOMYELITIS. Personali (La Semaine Médicale, 1902, No. 24).

This author has made use of plaster jackets in connection with suspension in the treatment of chronic meningomyelitis. It is his opinion that suspension will rupture all adhesions between the meninges and their adnexa and establish a sufficient flow of blood, and that the immobilization in the jacket will maintain the conditions of maximum extension and favorable circulation, so making sure the good effects of the suspension. The treatment is applied daily, the first suspension lasting only one minute, gradually increasing to four or five minutes, then remaining constant for about ten or twelve days. The jacket is put on during suspension as the body is then supposed to be in complete extension. Sayres' method is used and the jacket extends from the highest point possible to the iliac crests. The first jacket applied remains in position for three months, and during this time the patient is kept in bed, all organic functions being performed in this position. Walking or even sitting up is not permitted until the end of the third month. · The first jacket is then removed, suspension again employed and a second jacket placed in position in the same manner as the first. The patient is not now compelled to remain inactive, however, motion being possible as soon as the plaster has hardened. After two months the jacket is replaced by a system of carefully applied bandages, and after this has been in use for two months the treatment is considered complete. This method is largely protective in its results, and it is also necessary to remove all possible causes of disease, such as alcoholic excesses, etc., and to make use of tonic measures, such as subcutaneous injections of cocadylate of soda. Sixteen cases have been subjected to the foregoing measures by the author during the course of ten years, resulting in eight complete cures, four cases of marked improvement, four doubtful results, as complete treatment was not taken. Of these last two resulted later in failure. This treatment should be inaugurated early to obtain good results and is completely useless where there are manifestations of syphilis. JELLIFFE.

A CATATONIC CASE OF DEMENTIA PRAECOX. FR. MEENS (Psychologische en Neurologische Bladen, 1902, No. 1).

This author reports a case of a patient of English birth, twenty-two years old, of excellent parentage, and as far as could be ascertained no hereditary affection. The symptoms first appeared at the age of fourteen and at the age of fifteen melancholia and despondency increasing the patient was sent to Germany for treatment. During two years there was an increase of melancholia, hallucinations of persecutions, etc., with twitching of the nerves and misophobia. Although his mind continued clear he gradually lost self-control, the twitchings becoming more violent with an ever-increasing tendency to inflict injury upon his person. These injuries were all done with the right hand upon the left side, such as punching the left eye until the sight was entirely destroyed, striking the head against the bed and punching his side until a rib was fractured. With these fits of mutilation there were convulsive contractions of the muscles of face, throat, arms, trunk and abdomen. As self- control gave way there were violent stampings and screaming. He asserted his inability to repress these manifestations. The symptoms of touch disturbance were of a neurasthenic nature and there was neither hyperesthesia nor anesthesia nor analgesia which invariably occur in hysteria. He complained of a strange substance in his throat, of fever, toothache, etc.; he believed that the left half of his body was less developed than the right. There was a series of nervous tremors in close sympathy with these touch disturbances. The patient was given to self-abuse, a habit he contracted at an early age. He would frequently make violent efforts to expel the imaginary substance from his throat, or beat his jaw during toothache, and after urinating or stool passage he would pull the penis or force the rectum with such violence that the anus sometimes projected and bled and the scrotum was so pulled out that traumatic orchiepididymitis often occurred. The patient gradually grew very suspicious and lost all power of decision and interest in outside things, but he remained conscious of his condition, and unlike persons with mania for inflicting injury on themselves, he would complain of pain where he struck himself. He was well-formed and pleasant looking, head of normal form and measurement, face asymmetric, a little drawn on the left side, muscles well-developed, sound lungs, a hyperchlorhydric stomach, appetite whimsical, pronouncedly vegetarian, genital organs originally normal but terribly distorted by onanism. A gradual decline, when skin became atrophic and muscles decreased, was followed by death at the age of twenty-three. The writer was not satisfied with the diagnosis of "doubt mania" given this case. He was of the opinion that it was rather an unusual case of hebephrenia, a theory borne out by the age at which the first symptoms appeared, and the onanism, as well as the over-wrought mind, the melancholy and psychic delusions. Especially his unusual consciousness adds strength to the theory and throws light upon these catatonic movements which were of psychic origin. The second series of movements was related to his organic touch, but the third class did not seem to be capable of explanation, either by outsiders or the patient himself. It is the author's opinion that the spasmodic drawing of the muscles which caused the self-mutilations and twitchings was the effort of the nerves to get rid of the unbearable pressure on the brain which the patient described as a weight of 100 kilograms. The disassociation of the right arm, which always played the chief part in the mutilations, is also worthy of note in this connection. JELLIFFE.

TETANY AND MYOTONIA IN INFANCY. C. Hochsinger (Rev. Mens. des Maladies de l'Enfance, June, 1902).

The author holds that myotonia is a neurosis distinct from tetany, and the pseudo-tetanus of Escherich and belongs to the early period of infancy. Tetany in nursing children does not differ from the same manifestations in adults. Intermittent and tonic contractures, particularly in the muscles of feet and hands, with hyperexcitability of the motor nerves and the muscles are its main characteristics. The most important symptom of this hyperexcitability, which is the essential condition of tetany, is that of Chvostek, the sudden contraction obtained by striking branches of the facial nerve. In myotonia this reaction is not obtained. Trousseau's symptom is also present in tetany, consisting in the reproduction at will of the paroxysm, by pressing the affected part in the direction of its motor nerves or blood-vessels. Pressure on the nerves of the brachial plexus will in myotonia produce the contraction of the fingers, called the "phenomena of the fist," a sign seen only in infants six to eight weeks old. The fact that in early infancy the inhibitory centers presiding over the reflexes are not fully developed would account for this phenemenon without dependence on general hyperexcitability. True infantile tetany is developed solely on a rachitic foundation and is a neuro-muscular affection. As in rickets Kassowitz found that the largest number of cases developed in the early spring months, disappearing entirely at the end of summer. Myotonia, however, has no connection with rickets, developing in the course of gastro-intestinal intoxication it is not manifest at any particular season, except that the most serious intestinal disorders are likely to occur toward the end of summer. Myotonic contractions sometimes persist for weeks at a time, while those of tetany are paroxysmal and intermittent, in the former the attack comes on gradually, in the latter it is quite sudden. Myotonia exhibits no mechanical galvanic hyperexcitability of muscles or nerves and there is absence of facial symptom, neither is there evidence of the laryngo-spasms and the tonic and intermittent spasms of respiration usually occurring in tetany. Myotonia is manifest in the first weeks of infant life, while tetany usually appears at a later period. The contractions of tetanv have a tendency to recur, while in myotonia the attack always disappears with the cause. The spasms of myotonia are not affected by the phosphorus treatment which has shown good results in the neuro-muscular affections of infancy. JELLIFFE.

LATENT BRAIN ABSCESS BECOMING EVIDENT WITH SYMPTOMS OF ATROPINE POISONING. E. Aufrecht (Arch. f. klin. Med., Vol. 72, Nos. 5 and 6).

Being summoned to attend a case of supposed bronchial asthma in a laborer, the author administered atropine. This was followed by severe headache, twitchings, dilation of the pupils and marked confusion. These phenomena gave doubt as to the real diagnosis, especially as the prescription would appear to have been stronger than was intended. Five years previous the patient had received an injury to the head, and although he had then suffered from shock he had apparently made a complete recovery. The autopsy, however, revealed a large abscess in the left temporal region, which had never made its presence apparent during the five years since the injury had been received. JELLIFFE.

EPIDEMIC POLIOMYELITIS WITH REPORT OF TEN CASES. D. H. MacKenzie. Medical Record, October 4, 1902.

Thirty cases of poliomyelitis occurred near Poughkeepsie in 1899, in an epidemic resembling the extensive epidemics near Boston in 1894, and in Vermont during the same year. Of the ten cases reported by the author and seven others occurring in the same locality only two presented the classical symptoms of poliomyelitis. One case was undoubtedly cerebro-spinal meningitis, having the convulsions, coma and other symptoms. Three cases died, and in each the paralysis began in the lower extremities and extended upwards until the muscles of respiration became involved. Like the Vermont outbreak, while the clinical history of the majority of cases was typical of poliomyelitis, in some of them the features of peripheral neuritis were seen, while many of the fatal cases gave symptoms resembling cerebro-spinal meningitis. There is then presented the interesting fact that during the same epidemic and presumably from the same cause, there occurred cases of poliomyelitis, neuritis and cerebro-spinal meningitis. Certain mild cases of poliomyelitis in this epidemic terminated in complete recovery. Two fatal cases were diagnosed as Landry's paralysis, and reported in medical literature as such. From the fact that most of the cases were those of adults, from the pain, tenderness and paresthesia of the early symptoms, and from the fact that the paralysis extended from below upward, and also that all the cases which did not prove fatal in the acute stage except two fully recovered, we may conclude that a large percentage of these cases were really multiple neuritis. From the association in the same epidemic of cases of poliomyelitis, cerebro-spinal meningitis and multiple neuritis, the same microbic infection would seem to be present.

W. B. NOYES (New York).

HYSTERIA AFTER TRAUMATISM COMBINED WITH AN ORGANIC NERVOUS DISEASE. Meyer (Berliner klinische Wochenschrift, Aug. 4, 1902).

The author reports a case in which a man of forty-nine years, who has always been healthy, developed hysteria after an accident to his right elbow. He developed weakness of the extremities, paresthesia, shooting pains, tremor and a hesitation in speech. These symptoms varied in intensity, were irregular in nature and atypical in character. This fact with the general demeanor of the man led the author to suspect hysteria. The patient, however, also presented optic neuritis, Argyll-Robertson pupils and loss of knee-jerks. Meyer therefore concluded that the diagnosis consisted in an organic disease of the central nervous system combined with hysteria. He believed the organic condition to be locomotor ataxia or beginning progressive paralysis.

W. E. RATHE (Philadelphia).

ACROPARESTHESIA FOLLOWING TRAUMATISM. Sommer (Berliner klinische Wochenschrift, Oct. 6, 1902).

Sommer reviews the literature concerning acroparesthesia and reports two cases following injuries. Acroparesthesia depends upon an irritation of the vasomotor centers. The arteries become contracted and the nourishment of the nerve endings in the extremities is diminished. The main symptom is an unpleasant feeling in the hands, and more rarely in the feet. It is worse at night and morning. Occasionally these feelings change to acute pain. It is rarely limited to the peripheral distribution of any one nerve. The skin presents but slight or no disturbance in sensibility. There is no pain on pressure over the nerve, and no gross changes occur in motion. Its development is gradual. The etiology is not clear. This disease occurs most commonly in women at the menopause. Sommer advises that when paresthesia occurs after an injury, the possibility of acroparesthesia should be considered, and thus spare the patient the accusation of simulation.

W. E. Rathe (Philadelphia).

Myelorrhaphy followed by Return of Function. Stewart and Hart (Phil. Med. Jour., June 7, 1902).

These authors report the case of a young woman twenty-six years old, who had been shot, one of the bullets entering the spinal canal at the seventh dorsal vertebra, and causing complete abolition of motion and sensation below the level of the tenth dorsal spine. The operation, performed three hours after the shooting, exposed by incision and dissection that the right lamina of the seventh dorsal vertebra was crushed in, and the left lamina fractured at the base, while on removal of the spines and laminæ of the seventh and eighth dorsal vertebræ the cord was discovered to be completely divided, with a separation of three-quarters of an inch after the lacerated nervous tissue had been removed. The ends of the cord were drawn together by three chromicized gut sutures, one passing anteroposteriorly and the others transversely through the entire thickness of the cord. They were unable to approximate the dura. A small drain of gauze was left in place for twenty-four hours. The muscles were united by means of catgut, and silkworm gut was used to close the skin. After the operation the patient's condition was excellent, the wound healing by the seventh day. Sensation began to return in a few days, flexion of the foot was recognized after about two weeks, followed by powerful leg contraction, with consciousness of deep pressure over the limbs and abdomen. In three weeks pin-pricks could be felt as far as the umbilicus, although the patient could not localize the pain, urine could be felt in the bladder, but not voided, and the patellar reflex returned. After two months the knees could be fully bent, and after five the patient was able to slide out of bed into a chair without aid. The menses returned during the seventh month, and by the eighth good control over the bladder and bowels was regained, and she could stand without support. General improvement continued until in fourteen months in a tub bath she was able to feel water on the lower extremities and distinguish between hot and cold. In sixteen months she was capable of flexion and extension of the toes, legs and thighs, though flexion was stronger than extension. General health and sensation of temperature, pain and position were excellent. When the cord is divided or crushed, "no interference" is a good maxim for spinal surgery. There is special indication of myelorrhaphy when the cord has been cut in two by a sharp instrument or projectile, and it is the opinion of the investigators of this case that there should be immediate exploration to decide the exact nature of the injury and whether the symptoms are caused by pressure or by concussion.

Jelliffe.

# Book Reviews

NINTH ANNUAL REPORT OF THE BOARD OF MANAGERS OF THE CRAIG COLONY FOR EPILEPTICS.

THE yearly report of the Craig Colony, by its Medical Superintendent, Dr. William P. Spratling, have always been characterized by original data the value of which in the colonization of epileptics can hardly be overestimated. The present report, the ninth, is no exception to the rule, and presents an excellent summary of new facts not heretofore in evidence.

The past year's work has proven very satisfactory in that the Colony has discharged more patients as improved than in any previous year, while the death rate has been but 4 1-3 per cent.; some phase of epilepsy was the cause of death in 14 of the 33 cases. 182 patients were admitted for the year, 116 men and 66 women; the census, October 1, 1902, the close of the fiscal year, was 826. During the year 27 men and 11 women were discharged improved; 8 men and 3 women unimproved; while 12 were sent to state hospitals for the insane. A more careful selection resulted in a better class of cases being admitted for the year.

The desirability of a closer control of some patients who should remain at the Colony, but are not so inclined, is strongly advised as well as the necessity of all the houses for the colonists to be made thoroughly homelike in architecture and furnishing. The whole life should be simple and systematic; as strenuous life is not intended for the epileptic. A case illustrating the apparent direct transmission of the disease is graphically portrayed and the urgency of some legal restriction to marriage in the epileptic class is advocated. The futility and quackery of nerve nostrums is thoroughly discussed. The Colony is desirous to obtain samples of such preparations for analysis. The preponderating influence of heredity is given great prominence in the etiology of the disease; a combined heredity such as alcoholism, tuberculosis, epilepsy and insanity was proven definitely in 48 per cent. Provisions for the isolation of the tubercular and those suffering from the communicable diseases are asked for. The Craig Colony prize was given this year to Dr. Julius Donath for a paper upon "Cholin in Epilepsy and Its Significance in the Production of Convulsive Attacks." The educational, industrial and agricultural features of the Colony are excellently described and illustrated.

A special appropriation of $150,000 is asked for another year to provide accommodations for a large waiting list. The report is accompanied by a complete table of contents and an index. One happily misses the presence of dry-as-dust and meaningless statistics endlessly reiterated in so many annual reports of institutions. We are informed, however, that scientific data bearing on the nature and causation of epilepsy are kept in the medical records of the Colony, and may be obtained for study on application by those interested. The report in its entirety will be of great interest to both lay and professional readers who wish to learn of the progress of work in this wide field of modern philanthropy. The report is an example of what institutional reports should be. L. PIERCE CLARK.

ARBEITEN AUS DEM NEUROLOGISCHEN INSTITUTE AN DER WIENER UNIVERSITÄT. Herausgegeben von Prof. Dr. HEINRICH OBERSTEINER. IX Heft. Franz Deuticke, Leipzig und Wien.

In the past several volumes of Prof. Obersteiner's "Arbeiten" have been reviewed by the present writer, and occasion has been taken to speak in terms of praise of the work done in this institute. The latest volume is quite as commendable as any that has preceded it. It is composed of papers that are purely anatomical, or of papers that are clinico-anatomical. If less attention is paid to the former in this review, it is because papers of this character interest a smaller circle of readers, and also are difficult to describe in the space allotted to a review.

The ninth volume begins with two anatomical papers; the first is a study of the brain-stem of the *Delphinus delphis*, by R. Hatschek and H. Schlesinger, and the second a study of a brain from an Australian negro, by J. P. Karplus. The first paper represents a remarkably painstaking and time-consuming investigation.

O. P. Gerber and R. Matzenauer have studied a very interesting case in which leprosy and syringomeyelia were present in the same person, and it seems to be the first case of the kind on record. They do not jump to conclusions, and are cautious in what they have to say regarding the relation of the two diseases. Lepra bacilli were found in a piece of skin excised during the life of the patient, but none were found after his death in the peripheral nerves or spinal cord. These authors recognize that the absence of bacilli, or of histological changes in the nervous tissues, does not show convincingly that the leprosy had no causal relation to the syringomyelia, and further they mention that the very advanced age of the patient, over eighty, suggested an unusual cause for the syringomyelia. They, however, incline to the view that two diseases without intimate connection occurred in the same person, but they prefer to wait until other cases are reported in which a causal relation is more clearly shown before they give a positive decision regarding their own case. They also discuss the one-time important question of the relation of Morvan's disease to syringomyelia.

K. Tsiminakis describes a case of hypertrophy of the brain in a child eight years old. The brain without fluid in the ventricles weighed 1920 grammes. The hypertrophy affected all parts, and was not caused merely by proliferation of the neuroglia. It was a congenital condition. Chronic meningitis also was found.

R. Breuer and O. Marburg report two cases of apoplectiform bulbar paralysis, and then attempt to explain the symptoms observed in their two cases by the lesions found, and by a comparison with other cases in the literature. Disturbance of sensation in the face is attributed to degeneration of the spinal root of the trigeminus. They show that a slight degeneration of bulbar nuclei may exist without causing symptoms, and that paralysis of the soft palate, often bilateral with a predominance of the paralysis on the side of the lesion, is the result of implication of the middle or more cerebral portion of the nucleus ambiguus or its fibers, and that the facialis is scarcely concerned in this paralysis at all. The bilaterality is explained by partial decussation of fibers. From the study of a case the reviewer has recently made, he can support this opinion to the extent that the facialis is by no means the chief nerve to the soft palate. Breuer and Marburg conclude that deglutition and the motor innervation of the larynx depend on the integrity of the nucleus ambiguus and its fibers. The ptosis of one eyelid in association with myosis and retraction of the eyeball in their two cases is explained as sympathetic ophthalmoplegia, and they are led into a study of this condition, with the conclusion that sympathetic fibers pass through the internal capsule, decussate in the pons, and descend through the medulla oblongata to the spinal cord. A lesion of these fibers causes sympathetic ophthalmoplegia; on the opposite side if the lesion is at or above the optic thalamus; on the same side if the lesion is in the pons or medulla oblongata. A comparison of the cases of apoplectiform bulbar paralysis with one another shows that the most common symptom is contralateral dissociation of sensation with paralysis of deglutition and often aphonia or paralysis of the soft palate, and sympathetic ophthalmoplegia occurs only when the lesion is in the dorso-medial portion of the substantia reticularis lateralis.

In the paper by J. Tarasewitsch a study of the degeneration caused by a lesion of the optic thalamus and lenticular nucleus is presented. The direct ventrolateral pyramidal tract, first described by the reviewer, Tarasewitsch suggests may be merely the direct pyramidal tract in an abnormal position. This is improbable even though in his case the direct pyramidal tract was intact. In Tarasewitsch's case the lemniscus was relatively intact, but fornix fibers were much degenerated.

S. Kreuzfuchs has attempted in his paper to give the extent of the surface of the cerebellum.

R. Hatschek has a second anatomical paper in this volume; a comparative study of tegmental fibers and of the central trigeminus tract.

E. Zuckerkandl writes on the olfactory fibers of *Dasypus villosus.*

Acutely developing oculomotor palsy is not common, and the case reported by L. von Frankl-Hochwart is an important one. A man, who had had right-sided hemiplegia about nine months, observed suddenly while writing, a cloudiness before his right eye, and a half hour later he had diplopia, otherwise he was in good health. By evening of the same day he had right-sided ptosis. Death occurred twenty-eight days later. The nuclei of the oculomotor nerves appeared to be nearly normal. The oculomotor nerves were studied by the Marchi method, and the left was found to be normal, but acute interstitial neuritis was found in the right. An explanation for this peculiar finding is not offered, as there was no compression from an enlarged artery, and no meningitis, and signs and history of syphilis were wanting. The case shows very clearly that paralysis of the external ocular muscles may occur without paralysis of the internal ocular muscles when the lesion is neuritis and not in the nuclei of the oculomotor nerve.

By "symptom-complex of Benedikt" is understood hemiparesis with involuntary movement of the paretic side and crossed paralysis of the oculomotor nerve. In the paper by H. von Halban and M. Infeld the literature bearing on this subject is considered, and two cases, one with necropsy, are reported. The case with necropsy was as follows: A girl, aged fifteen years, fell on her head when she was ten months old, and became hemiplegic on the right side and had left-sided ophthalmoplegia. Later she had convulsions and hemichorea in the paralyzed limbs, and the right oculomotor and abducent nerves became paralyzed. She died from general tuberculosis. A calcified tubercle, having destroyed the nucleus ruber, was found in the left tegmentum. The trauma may have aided in the development of the tuberculosis. The lesions found did not fully explain the symptoms.

M. Schacherl describes the anatomy of the spinal cord of the *Myliobatis aquila.*

Prof. Obersteiner brings the volume to a close with an interesting anatomical paper. In a brain examined and described by him the lower olives to the naked eye, apparently, were absent. The clinical history could not be obtained. Microscopical examination showed that the pyramidal fibers extended on the outer side of each olive to the sulcus postolivaris. Obersteiner thinks that displacement of pyramidal fibers is not very uncommon, and he gives illustrations of variations in the position of these fibers in man and lower animals; in his opinion variations in these tracts are more common than in any other tracts. SPILLER.

Leitfaden der Elektrodiagnostik und Elektrotherapie für Praktiker und Studierende. Von Dr. Toby Cohn, Nervenarzt in Berlin. Zweite vermehrte und verbesserte Auflage. S. Karger, Berlin.

We have had occasion in speaking of the first edition of this work to commend it in no unmeasured words as a thoroughly practical work on the subject of electrodiagnosis and that the medical world at large has coincided with our opinion is evidenced by the appearance of the second edition within a comparatively short interval of time.

Electrotherapeutics has not remained in *statu quo* during this period and we are not disappointed in looking through this volume, for here we find that the new researches and conclusions, and practical devices of the last three years are incorporated.

The therapeutic portion in particular has been rewritten and enlarged. New special subjects, such as the Tesla stream and other new forms of electrical application have been incorporated, making the work the best of its kind.

The author has limited the portions bearing on pure physics, and we believe very wisely, to only that outline that is sufficient to understand the construction of the apparatus used. Muscle physiology is also well handled—concisely, scientifically and practically. We have seen authors of other works on the same subject lose themselves in the minutiæ and intricacies of muscle physiology—here not so. The major part of the work is devoted to the practical ends. This is what makes it so commendable. The mechanical construction of the book is excellent.

Jelliffe.

The Mattison Method in Morphinism. A Modern and Humane Treatment of the Morphine Disease. By J. B. Mattison, M.D. E. B. Treat & Company, New York.

This brochure of forty pages gives in detail the outlines of a form of treatment of the morphine disease which for years has proven helpful, since it has been constructed on sound principles and based on a fundamental knowledge of the conditions.

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Jelliffe.

The American Year Book of Medicine and Surgery. Under the General Editorial Charge of George M. Gould, M.D. Medicine. W. B. Saunders & Company, Philadelphia and London.

The year book comes this year with the same dress as last, in two volumes, making a more convenient book for reference than the older one bulky volume publication.

Such a publication as this should be, we believe, in the library of every practitioner who is at all interested in the scientific side of his calling, and of the many kinds of condensed literature of the day the year book still maintains its place in the front rank, exceeded in value by none and equalled by but one in the English language.

Dr. Archibald Church, of Chicago, retains the editorial supervision of the work in Nervous and Mental Diseases, and has given a most excellent résumé considering the space at his command. To be able to compress the more important features of the year's work in fifty pages, when the Neurological Jahrsbericht occupies over one thousand, is certainly a feat requiring no mean ability; and yet most of the important work has been included in neurology. Psychiatry has been neglected.

There is still too much of the patchwork character in the book and not enough working over of the material. Less clipping and more condensation of several articles into one would be advisable.

Brown.

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The Official Organ of

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The New York Neurological Society
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FOREIGN AND DOMESTIC AGENTS

S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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# Original Articles.

## REPORT OF TWO CASES OF BULLET INJURIES TO THE LEFT LATERAL HALF OF THE UPPER PORTION OF THE SPINAL CORD.[1]

By J. T. Eskridge, M.D.,
Denver, Colo.
CONSULTING ALIENIST AND NEUROLOGIST TO THE ARAPAHOE COUNTY HOSPITAL AND ALIENIST AND NEUROLOGIST TO ST. LUKE'S HOSPITAL.

AND

Edmund J. A. Rogers, M.D.,
Denver, Colo.
PROF. OF SURGERY, DENVER MEDICAL COLLEGE; SURGEON TO ST. LUKE'S HOSPITAL, ETC.

*(Case I.—Spiculae of bone driven into left side of sixth or seventh cervical segment; operation, death on the seventh day. Case II.—Compression of the left side of cord opposite the first and second costal segments by a 22-caliber bullet lying external to the dura; operation, recovery.)*

Case I.—Thomas E. W., white, single, *aet.* 31, gambler by occupation, born in Ohio, lived in Colorado since 1885, was admitted into St. Luke's Hospital February 23, 1898.

*Personal History.*—He had most of the common diseases of childhood, but apparently made complete recoveries from them. When he was 12 years old he ran a harrow pin through his left heel. A scar is now visible just behind the internal malleolus. He had typhoid fever when 19 years old and was sick for six or eight months. He has been a hard drinker for a number of years. He took the Keeley cure in 1893, and stopped drinking for about a year. Since 1894 he has taken from twenty to thirty drinks of whiskey each day. He has used a great deal of tobacco, both smoking and chewing, for a number of years. He has been addicted to indiscriminate and excessive sexual indulgence. He does not think he has ever suffered from any venereal disease. His family history, so far as he knows, is good.

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[1] This paper was prepared for the meeting of the American Neurological Association, June, 1902, by the late Dr. Eskridge in association with Dr. Rogers.

On February 21, 1898, he was in a saloon at Victor, Colorado, had an altercation with a man who pulled a gun on him, held it near his chin and fired. Every muscle seemed to relax, he fell to the ground, did not lose consciousness, but was unable to rise and his friends carried him home.

After having been placed in bed he complained of a numb sensation in entire left side from shoulder downward, and found that he was unable to move the left arm or leg. He was able to move the limbs on the right side. A bullet wound was found in the front of his neck just to the left of the median line. There was no wound of exit for the bullet. The wound in the neck was dressed. He was taken to Denver late on the evening of the 22nd, and placed in St. Luke's Hospital. At 11.30 A.M., of February 23, or about forty hours after the wound had been inflicted, I saw him in consultation with Dr. Rogers.

*Examination.*—He is perfectly conscious and able to answer questions. He gives a detailed and connected account of the altercation, the shooting, his feelings immediately after the wound was inflicted and during the time that has intervened since the injury. His statements in regard to his subjective sensations, and his answers to my questions during a prolonged examination, lasting about one and a half hours, appear reliable and correct.

He is a large, muscular man, weighing about 200 pounds. His appearance shows the results of alcohol and other dissipation. He complains of shortness of breath, weakness, and pain in the right upper arm. The left leg and arm are completely paralyzed. He can move the right leg and arm. The left leg and arm feel numb, as if they were "asleep." This sensation is most marked in the hand. All the deep reflexes are normal in right leg, but absent in left. Plantar reflexes: R., present; L., absent. Cremaster reflex: R., present; L., absent. Abdominal reflexes absent. Deep reflexes of arms: R., present; L., nearly absent. Dyn. R., 62; L., 0. The face and tongue are unaffected. The neck muscles are not tested on account of the patient's weak condition and shortness of breath.

*Tactile sense* is lessened but not abolished on the left shoulder and over the left upper arm nearly to the elbow and to a very slight extent over the right hand and wrist. Throughout the trunk and other portions of the limbs, tactile sense appears to be unaffected.

*Pain sensation* is absent throughout right side from first intercostal space downward. It is absent also in the right arm from about the elbow downward in front and over the posterior surface of the entire arm. It is absent over the left shoulder and upper arm front and back downward nearly to the elbow. Over all other portions of the body pain sensation appears to be normal.

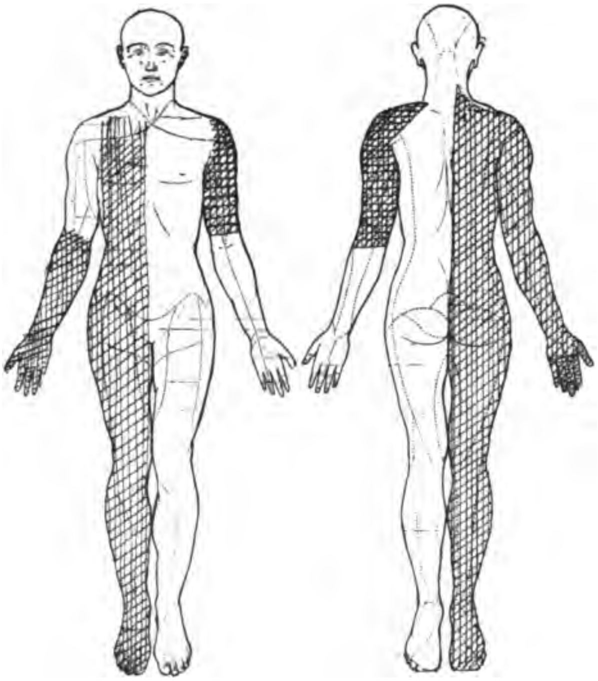
*Temperature sensation* throughout the areas in which pain sensation is lost, cold and warm substances are spoken of as warm. He is unable to distinguish between hot and cold or cool and warm substances in any portion of the analgesic areas. While the sensation of heat caused either by warm or cold substances cannot be said to be lost, yet the power to distinguish between substances of different temperatures is completely lost. Except in the areas above mentioned, temperature sensation does not vary from the normal.

Posture or muscular sensation seems normal in right leg and arm, but apparently lost in left leg and arm. Joint sensation seems perfect. Pressure sensation is imperfect in the tactile anesthetic areas, otherwise it is normal. Taste, smell and hearing seem normal. Vision, fields, disks, and fundi present no evidence of lesion. There is slight ptosis of the left upper eyelid. The right pupil is twice the size of the left; the latter is apparently normal in size. Both pupils respond to light, shade and accommodation, but the right is less active than the left. The patient was not aware that any difference in the size of the pupils had existed prior to the injury.

*Diagnosis as to the Character, Extent and Location of the Cord Lesion.*—It was of considerable importance to solve these problems, if possible, before attempting to operate. The wound had been carefully dressed by Dr. Rogers before I saw the man. I was informed that a 38-caliber bullet had penetrated the anterior portion of the neck nearly in the median line and apparently passed nearly directly backward. I did not see the wound until fifty-three hours later.

The results of the examination showed that only the left side of the cord had been injured and that the destruction of this side was not complete was evident from the fact that most of the fibers of tactile sense seemed to be intact. That the pyramidal fibers had been completely cut across was evidenced from the fact that all reflexes, both deep and superficial, were absolutely abolished on the left side of the trunk and in the left leg, and were scarcely perceptible in the left arm, forty hours after the injury had been received, although the corresponding reflexes on the right side, except the abdominal, were present and slightly increased. The fibers for muscular, pain and temperature sensations had also been cut across at the seat of the injury on the left side.

The questions were debated: Was the cord injury due directly to the bullet passing through the cord and membranes, or to a spicula of bone having been driven through the membranes into the cord by the force of the bullet? That a fatal hemorrhage had not occurred led me to believe that the bullet had not plowed its way through the membranes and cord. That the injury to the left side of the cord was not due to the presence of the bullet in the spinal canal and its pressure upon the cord seemed certain from the fact that the pyramidal tract had been completely cut across, as shown by the absence of the reflexes on the left side. It seemed to me most likely that the cord had been injured by some sharp cutting substance, probably a spicula of bone. If I were correct the probability was that the spicula of bone was partially or completely imbedded in the substance of the cord. Accordingly an operation was recommended.



Figs. 1 and 2—Horizontal lines indicate loss of tactile sense. Vertical lines indicate loss of pain sense. Oblique lines indicate loss of temperature sense.

The injury did not involve the whole of one lateral half of the cord, else the fibers of tactile sense would have been destroyed and we would have had tactile anesthesia on the right side. It must be remembered that tactile sense was not lost in any portion of the body or limbs, but was lessened in portions marked by horizontal lines in Figs. I and II.

As near as could be determined, the lesion was located in the sixth or seventh cervical segment of the cord.

The bullet had not been recovered, but where was it? Probably lodged among the spinous processes of one of the cervical vertebræ. If this were the case an operation for its removal at that time might endanger the man's life from hemorrhage, especially in his prostrated condition from shock, shortness of breath and refusal to take a sufficient quantity of food. On the other hand, if the injury to the cord were due to the presence of a spicula of bone in it, the sooner the offending substance was removed, the better.

The patient has to be catheterized. His temperature in each axilla is 99.2° F.; pulse 110; respirations 20 when he is quiet, but the latter increases considerably on the slightest exertion. He is restless, feels miserable and it is with great difficulty that we can get him to take sufficient liquid nourishment. The heart is free from murmur, the kidneys are acting well and the urine shows neither albumin nor sugar. The extreme restlessness and the poor quality of the pulse made it desirable to avoid a serious operation at the time, if possible. It was decided to nourish the man as much as possible and to stimulate him, to see if we could get him in a better condition for an operation.

February 24—He shows some apparent improvement; the temperature is normal, the pulse 80, quality a little better, respiration 20.

February 25—It is evident that the improvement is only temporary; the temperature begins to rise 99° F. at noon; the pulse and respiration, while not increased in frequency, are not as good as they were the day before. The patient is becoming apathetic. The serious nature of his condition is explained to him. He is told that an operation may do good, but that the chances are against him. He decides that his physicians are the best judges and leaves the matter of operating to them.

### OPERATION AND REMARKS.

BY DR. ROGERS.

Operation at 4.15 P.M., on February 25. A 38-caliber bullet is extracted from beneath the skin on the posterior portion of the neck, about one inch to the left of the median line. It is found by passing the finger in the track made by the bullet posterior to the spinal column that the sinus leads from the transverse process of what is apparently the fifth cervical vertebra. The conical end of the bullet is found to be flattened, its sides scarred, a piece of the metal clipped off, and left as was supposed in contact with the bone. On enlarging the sinus made by the bullet in its track through the bones of the spinal column, it is ascertained that the bullet emerged from the bone through the fossa between the fourth and fifth cervical transverse processes. The lamina of the fifth cervical vertebra is quite loose on the left side and broken into many pieces. The bullet had entered the spinal canal through the body of the fifth cervical vertebra. A piece of bone ⅜ of an inch in diameter is found within the spinal canal and lying external to the membranes. Two smaller pieces of bone, one round, 3-16 of an inch in diameter, the other a thin spicula, about ⅛ of an inch in diameter, had been driven through the membranes and into the lateral substance of the cord. These pieces of bone are removed. The bleeding is quite free. The bullet had not torn the membranes and so far as we were able to determine, it had not directly injured the cord. The cord was apparently damaged solely by the small pieces of bone that had been driven into its substance. There is not much bleeding within the membranes.

The patient is returned to the surgical ward at 5.30 P.M. During the night his temperature did not fall below 98° F., nor rise above 98.2° F. The pulse immediately after the operation was 110, rose to 116 by 7.30 P.M., but was 96 at 5 the next morning. Respirations continued about the same as before the operation, 24 per minute. It was evident that the operation caused no perceptible shock or apparent depression.

By 10 A.M., of the 26th, T. 101.4° F.; P. 108; R. 30. From this time on the decline was progressive and comparatively rapid. At 12 M., T. 102° F.; P. 132; R. 32. At 6 P.M., T. 102.2° F.; P. 140; R. 39. During the night the pulse ranged from 140 to 150, respirations from 36 to 40 and the temperature at 6 A.M. of the 27th was 103.6° F. At this time he had become comatose. He died at 12.30 P.M. on the 28th. His temperature did not go above 103.4° F.

Autopsy on the body of Mr. Thomas E. W. was made at 4.30 P.M. of February 28, 1898, four hours after death. The body is still warm; post-mortem rigidity is just beginning in jaw, chest and limb muscles. Only the parts at the site of the injury and surgical operation were examined. No pus and very little blood was found. The cervical portion of the cord was greatly softened. This softening, inflammatory in character, had begun to involve the medulla. On sectioning the cord into blocks the edges at the end of each section everted, especially was this well marked opposite the seat of injury to the cord. The substance of the cord at this point seemed to have undergone softening throughout its entire transverse extent.

The wound of the cord inflicted by the spiculæ of bone was in the left lateral aspect of the cord and did not appear to penetrate more than one-half the distance from the surface to the central canal of the cord. The wound involved directly only the extreme outer portion of the gray matter. The wound was wedge-shaped, with its base at the surface of the cord. The spiculæ of bone had not penetrated the anterior and posterior portions of the left lateral half of the cord, neither had the central gray matter of this half of the cord been reached by the spiculæ.

The cervical and upper costal portions of the cord and the medulla were placed in Müller's fluid and allowed to harden slowly for three months. The fluid was poured off and renewed every few days during the first two weeks. Before the cord was subjected to microscopic examination it was placed in a 2 per cent. solution of formaldehyde for a few days.

The microscopical examination was made by the late Dr. E. R. Axtell. Over 100 sections from the medulla, cervical and costal portions of the cord were carefully cut and examined. The sections showed softening throughout the cervical region of the cord. The softening was slight in the right lateral half of the cord, but in the left, especially in the region of the injury inflicted by the bone, it was very well marked and attended by hemorrhagic extravasation. The microscopic sections developed the fact that the spiculæ of bone had not penetrated the left lateral half of the cord to more than one-third the distance to the central gray matter immediately surrounding the central canal of the cord. The anterior and posterior portions of the left half of the cord had not been injured by the bone. The left side of the cord immediately in the vicinity of the lesion in the cord made by the pieces of bone, showed a degree of softening further advanced than the parts more remote from the site of the injury.

*Case II.*—Wm. R. D., *aet.* nineteen years, white, male, born in Texas, by occupation laborer in a cotton mill, was admitted into the surgical ward of the Arapahoe County Hospital, March 6, 1898, about 7 P.M. The family history was negative. The boy was tall (height 6 ft. 2 in.), lean and lanky. He was not otherwise well developed. His personal history was uninteresting.

On March 6, 1898, he was accidentally shot by a companion who stood some fifteen or twenty yards behind him. A 22-caliber, short conical bullet, coming from a Colt rifle, struck him in the left posterior portion of the neck. He immediately fell to the ground, but consciousness was undisturbed, and he was able to describe subsequently all the sensations that he experienced at the time. His companions found that he was paralyzed in the left leg and to a partial extent in the left arm. Immediately after the receipt of the injury the boy experienced a numb sensation in the right leg and arm, and great pain in both arms and legs. The pain was more severe in the limbs on the left side than in those on the right.

I saw the patient at 9 P.M., five hours after the accident, in consultation with Drs. Rogers and Howell T. Pershing, the former one of the surgeons and the latter the neurologist, to the hospital. I wish in this connection to acknowledge my indebtedness and express my thanks to Drs. Pershing and Rogers for the privilege of studying the case over a prolonged period and for permission to use my notes of it in connection with Case I, just reported in this paper.

The first neurological examination was made conjointly by Dr. Pershing and myself. All the muscles of the left leg are very weak, yet by an effort of the will almost all movements can be effected, although very feebly. Complete paralysis of the left wrist and fingers exists. He cannot extend the left arm at the elbow, but can feebly flex it at this joint. None of the shoulder movements are entirely lost, but are all very weak. All muscles of the right leg, arm, sides of the neck, face and tongue are normal in strength. Dyn. R. 130; L. 0. There is no ptosis of the upper eyelids and the pupils are equal in size and respond readily to light, shade and accommodation. Deep reflexes of legs and arms are slightly increased on right side and decidedly increased on the left. Plantar and cremaster reflexes: R. normal; L. greatly diminished. Abdominal: R. present; L. absent.

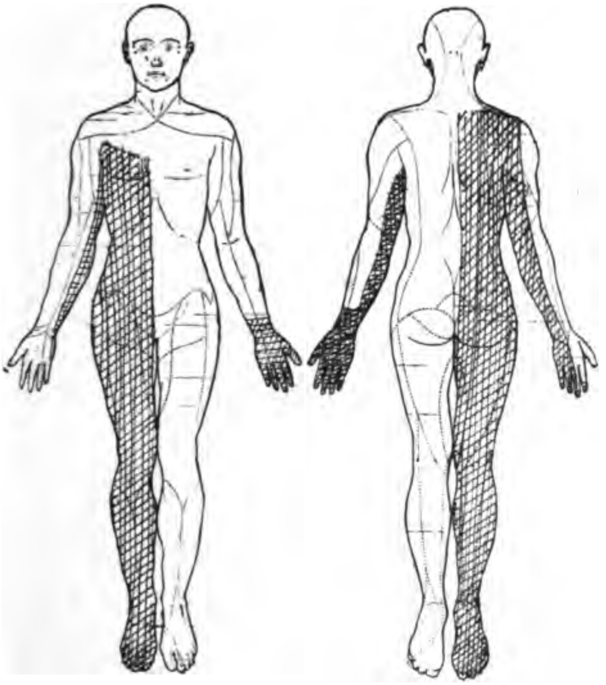
*Sensory phenomena:* Tactile sense is partially lost on the entire left hand, on the anterior surface of forearm for a distance of two inches above the wrist joint, on the posterior surface of forearm to a point three inches above the wrist joint, and on the posterior and extreme inner surface of the forearm and arm nearly to the axilla. (See Fig. IV.) Over all other portions of the body and limbs tactile sense is normal.

*Pain sensation* is lost in the entire right leg, on the right side of the trunk anteriorly to the third intercostal space and posteriorly to the seventh cervical spine; in the right arm and hand anteriorly there is only a slight disturbance on the extreme inner margin, posteriorly on the hand and arm to the shoulder on the inner one-half of the surface; over left hand and arm anteriorly, there is apparently no disturbance to pain sensation, but posteriorly marked loss in the tactile anesthetic area.

*Temperature sensation* is perverted or lost in the areas in which pain sensation is affected. (See Figs. III and IV.)

*Muscular sense* is absent in left leg and hand. Pressure sense absent in area of tactile anesthesia. Joint sense is absent in left fingers and wrist.

Ten days after Dr. Rogers and I had studied Case I, we encountered Case II, almost a counterpart, so far as neurological symptoms were concerned, of Case I, except that in Case II, the lesion was a little lower in the vertical level of the cord, and the deep reflexes were preserved on the left side and slightly increased. It will be remembered that loss of pain and temperature sensations began opposite the first intercostal space in Case I, and opposite the third intercostal space in Case II. In Case I all the reflexes, deep and superficial, were abolished on the left side from the level of the lesion in the cord downward, just the opposite of what we have just stated occurred in Case II.



Figs. 3 and 4—Horizontal lines indicate tactile anesthesia. Vertical lines indicate analgesia. Oblique lines indicate loss of temperature sensation.

Having so recently met with Case I, studied the clinical phenomena and examined the lesion in the cord at the post-mortem examination, I felt justified in making a diagnosis of pressure on the left lateral half of the cord opposite the second or third costal segment. In the light of my recent experience with Case I, Dr. Pershing readily agreed with me in the main features of the neurological diagnosis. It seemed to both of us a most favorable case for immediate surgical interference, and the patient agreeing, Dr. Rogers at once proceeded to operate.

### OPERATION AND REMARKS.

BY DR. ROGERS.

The wound in the skin, indicating the point at which the bullet had entered the neck, was small and situated a little to the left of the median line and at a point nearly opposite or a little below the spinous process of the seventh cervical spine. On following the track of the bullet from its point of entrance posteriorly, the bullet and a small spicula of bone are found within the spinal canal, external to the membranes, but in such a position as to allow them to exert pressure on the left lateral half of the cord at a point opposite its upper costal region, or it may be opposite the junction of the cervical and costal regions. It is impossible to determine upon which segments of the cord the bullet presses. The spinal membranes had not been injured by the bullet or spicula of bone. There is very little hemorrhage in the spinal canal. Only a small quantity of blood is lost during the operation.

The wound healed satisfactorily and surgical recovery was rapid and uninterrupted, the temperature at no time rising more than one or two degrees above normal. The pulse and respiration were only slightly affected.

### SUBSEQUENT NOTES BY DR. ESKRIDGE.

On March 12, six days after the receipt of the injury and the surgical operation, the sensory phenomena remained about the same as they had been before the operation, but the boy seemed greatly depressed. The temperature and respiration are nearly normal, but the pulse is weak and 100 per minute. The appetite is poor, the patient restless. It is difficult to get him to make any muscular movements. Both knee-jerks are absent. The other deep reflexes are lessened and all the superficial reflexes are absent. The right pupil is nearly twice as large as the left.

March 16, 1898. Right leg is strong in all movements. Left foot in almost complete plantar flexion, but he is able by voluntary effort to increase the plantar flexion of left foot. Dorsal flexion of the left foot completely lost. There is slight power of movement at left hip and knee. Dyn. R. 30; L. 0. Right elbow and shoulder movements about one-half normal strength. Unable to flex fingers of left hand, can extend left hand at wrist. He can flex left arm at elbow, but cannot extend it. Shoulder movements lost. Intercostal muscles of left side paralyzed. The major pectoral muscle on each side acts well.

Knee-jerks, R. normal; L. lessened. Plantar; R. slight, L. absent. Tendo Achillis, R. slight; L. absent. Cremaster and abdominal reflexes present. Deep reflexes of arms increased, the left to a greater degree than the right. The strength of the muscles about neck and shoulder, for the most part, cannot be tested on account of the presence of the dressings.

Sensory phenomena. Tactile sense throughout the entire right side to a camel-hair pencil held in contact with the parts is present, and also throughout left side, except over a small area on lower posterior inner one-half of upper arm, one inch in width and four inches long; over another area on forearm extending from elbow to wrist joint, one inch in width and lying on the extreme posterior ulnar of arm; also over entire left hand, palmar and dorsal surfaces and for about three inches above the wrist both anteriorly and posteriorly.

Temperature and pain sensations absent on right side from the third intercostal space downward, also over areas of tactile anesthesia and dulled over portions of right forearm. Posture sense absent in left arm and leg. Joint sense absent in left wrist and finger joints; present throughout right side. Examination of the organs of special sense reveals nothing abnormal except that the right pupil remains about twice as large as the left. Both responsive, left more readily than the right.

March 19. *Slight improvement.*

March 24. All movements of left leg, including foot and ankle have returned, but they are weak. No improvement in left arm movements, except that he can rotate the left forearm slightly. A few days later he could flex and extend hand at wrist. All forms of sensation remain about the same.

April 8. Tactile sense is restored on left wrist and hand down to the metacarpo-phalangeal joints, except on radial side of hand over which loss of tactile sense still remains sufficient to be detected by a camel-hair pencil in motion. Thermo-anesthesia is lessening on right arm and foot, that on the leg and body remain nearly the same as at first. Loss of pain sensation is not lessened but has extended a little on posterior surface of right arm. Ankle-clonus is recognized on the right side for the first time today. Deep reflexes exaggerated. Muscular sense still absent in left arm and leg.

He gradually improved, and by May 1, was able to walk with the assistance of a cane. When he first began to try to walk it was extremely difficult for him to do so on account of the loss of muscular sense in the left leg. He had almost completely recovered by July 1, except that muscular sense in the left leg, and arm is still defective. He left the hospital early in August. At that time he was feeling quite well, muscular sense was lessened in left arm and very decidedly defective in left leg, requiring him to use his eyes in walking. He had not entirely recovered his normal power of motion in the left leg and arm. Pain and temperature sensations were less acute on the right side than on the left. Tactile sense seemed perfect everywhere. I saw him one or two months later and his recovery was apparently complete, with the single exception that muscular sense was still defective in left leg, but much less than when he left the hospital. In the fall of 1898 he went to Nebraska and resumed his work in a cotton mill. I have not been able to obtain any word from him since he left Colorado, although I have written two or three times.

It is almost enough to make one weep to be apparently so near the solution of interesting problems, determining the function of the direct cerebellar tracts and the position in the cord of the fibers for pain, temperature and muscular sensations, and utterly to fail.

The spiculæ of bone had apparently completely cut across the left direct cerebellar and the crossed pyramidal tracts and the posterior portions of the antero-lateral ascending tract and the anterior ground fibers. Had the lesion extended no further than that directly inflicted by the spiculæ of bone, and had the patient survived a few weeks after the injury, a careful study of Case I would in all probability have resulted in brilliant results. The early death of the patient, the extensive inflammatory softening and the hemorrhagic infiltration blasted my hopes. My experience seems, in this direction, to be but a repetion of that of all neurologists who have had an opportunity to study similar cases. Sir William R. Gowers in 1878 recorded a case in many respects, so far as the lesion is concerned, almost a complement to Case I, detailed in this paper. In this hemorrhage and hemorrhagic extravasation took place[2].

There are, however, several points of interest suggested by the report of the two cases included in this paper, and seem to me worthy of discussion by the members of this Association. The ciliospinal center was affected in both cases. In each, it will be remembered, the injury was to the left side of the cord. In Case I, I quote from notes, made at the bedside of the pa-

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[2] "Diseases of the Nervous System," Vol. I. Third Edition, 1899, p. 235. Blakiston Son & Co., Philadelphia.

tient, "There is slight ptosis of the left upper eyelid. The right pupil is twice the size of the left; the latter is apparently normal in size. Both pupils respond to light, shade and accommodation, but the right is less active than the left. The patient is not aware that any difference in the size of the pupils existed prior to the injury." It may be well for me to remind you in this connection that the injury to the cord in Case I was to the sixth and seventh cervical segments. In Case II, the bullet lay in the spinal canal, external to the dura, in such a position as to exert pressure on the second and third costal segments. In this case there was neither ptosis of the upper eyelid nor affection of either pupil a few hours after the receipt of the injury; and dilatation of the right pupil was not observed until six days after the receipt of the injury, although the boy was visited by me daily and observed closely. By this time edema of the cord had probably taken place and involved the first costal segment. On March 16, the right pupil remained about twice the size of the left. On March 19, both pupils were equal and remained so until the patient passed from under my observation, several months later.

I was unable to satisfy myself that the palpebral fissures varied from the normal either in Case I or II.

The results obtained by a careful study of the two cases reported in this paper suggest that there may be fibers in the cervical region of the spinal cord that aid in elevating the upper lid of the eye of the same side, and in modifying the size of the pupil of the opposite side. I know that these results are directly opposite to what has been found by experimentation, but are not the conditions different? In experimentation the nerve roots have been irritated or sectioned. In most cases of disease of the cervical or extreme upper costal regions of the spinal cord attended by pupillary changes the nerve roots have been affected, besides there have been, in most instances, vasomotor disturbances. In neither case reported in this paper were there any vasomotor changes on either side. In Case II, the irritation apparently never extended to the nerve roots. In Case I there was no evidence that it had at the time of the examination, such as would have been shown by vasomotor disturbance.

Irritation of the nerve roots of the cervico-brachial plexus, certainly of the lower portion will give rise to dilatation of the pupil and widening of the palpebral fissure of the same side. Seguin observed myosis after section of these roots.

At first I thought my observations were at fault. I went over them repeatedly and the results were always the same. In Case I, the left pupil was normal in size and responded readily to light, shade and accommodation. The right was twice the size of the left and reacted slowly and imperfectly to all tests. In Case II, the pupils at first were equal and normal in size and both reacted perfectly. Six days later the right pupil was twice the size of the left and like the right pupil in Case I, reacted tardily and imperfectly. This condition continued for a period of seven days. At the expiration of this time the right pupil was as small as the left had been from the first and responded readily and perfectly to all tests. After all, we should expect the fibers for the ciliospinal center to cross in the medulla, but so far I have been unable to find any recorded cases that seem to confirm this. Kocher believes that the ciliospinal center extends from the medulla to the first costal segment.

One of the most curious conditions in Case I, was the ptosis of the upper eyelid on the side of injury to the cord. The patient said he had, so far as he was aware, no venereal disease and he was not a man likely to deny syphilitic infection had he suffered from it. I was unable to find any other symptom pointing to syphilis. His physician had never observed anything wrong with the man's eyes prior to the injury from the bullet.

It is interesting to note in connection with Case I, that all reflexes were abolished from the level of the cord injury downward on the side of the lesion, and on the side of the body on which the only form of sensation abolished or disturbed was muscular sensation. This not only supports Bastian's theory that a complete severance of the cord in any portion abolishes all reflexes below the point of lesion, but that so far as the cord is concerned the presence of the reflexes is dependent upon the motor and muscular sensation fibers and their associated neurones, axons and dendrons.

That some of the fibers of muscular sensation run in the posterior median columns seems certain from a study of the symptoms and pathology of tabes. In Case I, these columns were not affected by the lesion, yet loss of muscular sensation was complete on the side of the injury. The inference naturally is, that all the fibers of muscular sensation do not run in the posterior median columns. In this connection a quotation from Gowers is interesting. "The function of the direct cerebellar tract has still to be demonstrated. There are certain resemblances between its fiber-relations and those of the posterior median columns which gives countenance to Flechsig's theory that it conveys impressions from the muscles of the lower part of the trunk and between the trunk and the lower limbs. But the origin of its fibers from the cells of the posterior vesicular column, and the mystery attaching to the latter, give additional obscurity to this structure. The fine nerve-plexus between the cells seems connected with the fibers of the posterior roots, and this plexus is said to atrophy early in locomotor ataxy. The obscurity will, indeed, be dispelled if we can accept the speculation of Sherrington that this cylinder is really part of the series of ganglia on the posterior nerve-roots, which occupies a position within the spinal cord, and is continuous instead of broken into ganglia[3]."

Unfortunately a study of Cases I and II gives us no clue to the exact positions in the cord occupied by the fibers for pain and temperature sensations and tactile sense further than to emphasize the fact already known that the fibers for pain and temperature sensations lie near together in the cord and are not immediately joined by the fibers for tactile sense.

Pressure sensation is closely related to tactile sense and is probably a part of it. In the cases reported in this paper pressure sensation was only involved in the areas in which tactile sense was affected. The power of localization seemed to be preserved except in these areas.

What is joint sensation? With what form of sensation is it associated? Is it a part of tactile sense? In the above cases it was preserved, as nearly as I could determine, except in the joints over which the skin was completely or partially anesthetic.

The interesting character of the cases that I have just reported and the care with which I have tried to study them, constitute my only excuse for the length of my paper.

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[3] *loc. cit.*, p. 237.

# A STUDY OF SENSATIONS IN MOTOR PARALYSIS OF CEREBRAL ORIGIN BASED UPON THIRTY-FIVE CASES.*

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The question concerning the relationship of motor and sensory localizations is still unsettled, notwithstanding the considerable number of data accumulated in the literature. The doctrine of the sensori-motor area as one localization, as well as the doctrine of localizations separate for motor and sensory functions, is ardently defended by equally prominent authorities on either side. The subject as it stands now, although far from being solved, is nevertheless so far advanced that conclusions regarding one of the two functions cannot be drawn, unless a study of the other is made at the same time.

Disturbed functions, motor and sensory, of cerebral origin, may be due to cortical, subcortical, capsular, peduncular, or pontine lesions. The problem of localizations is therefore vast; its solution is as yet surrounded with a mystery.

The author of the present contribution attempted to approach one portion of this difficult problem, and while he does not pretend to solve the question in its entirety for want of pathological material, at least basing himself upon facts demonstrates with evidence, the clinical aspect of one of the views held by one of the two schools.

The study of the subject was undertaken by the writer without any preconceived idea, and without the slightest inclination towards one or the other view held by authorities in this country or abroad. His aim was to determine in the most impartial manner the condition of sensations in motor paralysis of cerebral origin.

The thirty-five cases were examined with the utmost care: selection was made of cases with perfect, or good mental lucidity, so that the question put to them be correctly answered. Cases of doubtful nature were not taken in the series. Also were excluded those cases which in addition to the motor cerebral symptoms presented symptoms of spinal diseases or of local affections.

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*Read before the Philadelphia Neurological Society, October 28, 1902.

Each case was examined repeatedly on the same and the following day. Considerable time was spent in repeated attempts to mislead the patient in order to verify the answers; the results were uniform. Finally a reëxamination of all the cases was made a month later and to my great surprise the data collected proved to be exactly the same as at the first examination.

One word more about my method: Every case was examined in regard to hysteria, and in order to attribute the sensory disturbances to their real cause, all cases with hysterical stigmata, except one, were excluded.

### SUMMARY OF THIRTY-FIVE CASES OF HEMIPLEGIA STUDIED FOR DISTURBANCES OF SENSATION AT THE PHILADELPHIA HOSPITAL AND JEFFERSON COLLEGE HOSPITAL.

Case I, No. 12. Infirmary. John Veasey, aged seventy-six. Complete right hemiplegia of one and a half years' duration. Movement in leg better than in arm.

Sensations: Upper limb—Touch anesthesia, which disappears at the root of the limb; pain, analgesia from fingers to elbow; to heat anesthesia, to cold anesthesia from fingers to elbow, above, hypesthesia.

Trunk—Touch normal, pain hypalgesia, to heat hypesthesia, to cold hypesthesia.

Lower limb—Touch anesthesia; pain, hyperesthesia from toes to knee, above hypesthesia; to heat hypesthesia, to cold hypesthesia.

Face—Touch normal, pain analgesia, to heat hypesthesia, to cold hypesthesia.

Astereognosis is complete. Position of upper limb is normal, special senses are normal, no paresthesia.

Case II, No. 5. Infirmary. Joseph Laselle, seventy-three years old. Complete right hemiplegia a year ago. Arm in contracture. Some movements in the leg.

Sensations: Upper extremity—touch anesthesia, pain hypalgesia on all aspects of the limb. From the fingers up hypalgesia becomes less marked towards the root of the limb where pain is normal. Heat not felt at all, to cold hyperesthesia.

Trunk—Touch anesthesia, pain hypalgesia. To heat normal, to cold hyperesthesia.

Lower extremity—Touch anesthesia, pain hypalgesia. To heat anesthesia, to cold hyperesthesia.

Face—Touch normal, pain analgesia. To heat normal, to cold hyperesthesia.

Astereognosis is complete. Position of paralyzed arm: patient cannot find the paralyzed limb. Special senses: hearing impaired on the right side. Paresthesia present.

Case III, No. 10. Infirmary. Gottlieb Schneider, sixty years of age, shoemaker. Complete left hemiplegia of seven years' duration. Some movements present, more in arm than in leg.

Sensation: Upper limb—Touch normal, pain hypalgesia (slight); pain becomes normal at the root of the limb. Heat taken for cold, to cold normal.

Lower limb—Touch normal, pain hyperalgesia, heat taken for cold, to cold normal.

Patient states that he was totally anesthetic to all sensations for a long time, but they gradually returned when motion improved.

Trunk—Touch normal, pain hypalgesia, to heat hypesthesia, to cold hyperesthesia.

Face—Pain, touch and temperature normal.

Astereognosis for small objects is present. Position of paralyzed limbs can be determined with the normal limbs. Special senses are normal, no paresthesia.

Case IV, No. 17. Infirmary. Frank Morbock, aged fifty-three years, laborer. Complete right hemiplegia with some impairment of speech of six months' duration. Motion is better in the lower than in upper limb.

Sensations: Upper limb—Touch normal, pain hypalgesia (this is equal at the end and root of the limb); to heat hypesthesia (becomes normal at the root of the limb), to cold hypesthesia.

Trunk—Touch normal, pain normal, to heat hypesthesia, to cold normal.

Face—Pain, touch and temperature normal.

Astereognosis is present for small objects; cannot recognize size and material of an object. Position of arm is not easily recognized with the normal limb. No paresthesia. Special senses normal.

Case V, No. 30. Infirmary. Michael Mooney, about seventy-five years of age, plasterer. Complete right hemiplegia of one year's duration. Some movement in the arm, but none in leg.

Sensations: Upper limb—Touch hypesthesia (slight), pain hypalgesia (slight), to heat hypesthesia, to cold hypesthesia (slight).

Trunk—Touch hypesthesia, pain normal, to heat hypesthesia, to cold normal.

Lower limb—Touch hypesthesia and delay, pain hypalgesia, to heat anesthesia, to cold hypesthesia from toes to knee, above, normal.

Face—Touch normal, pain hypalgesia, to heat hypesthesia, to cold normal.

Astereognosis absent. Paresthesia absent. Special senses: Hearing is equally impaired on both sides. Left pupil is larger than right.

Case VI, No. 15. Infirmary. Herman Schmidt, aged sixty years, machinist. Complete left hemiplegia of eleven months' duration. Arm and leg almost entirely without movements.

Sensations: Upper limb—Touch hypesthesia, pain, delay from fingers to wrist; above, analgesia. To heat anesthesia, to cold anesthesia.

Trunk—Touch normal, pain analgesia in some areas, hypalgesia in others. To heat anesthesia, to cold hypesthesia.

Lower limb—Touch normal, pain hypalgesia and delay, to heat anesthesia, to cold hypesthesia.

Face—Touch hypesthesia and delay, pain normal, heat taken for cold, to cold normal.

Astereognosis is present for every form. Paresthesia present (cramp-like sensation and numbness). Special senses: Hearing lost on paralyzed side, left pupil sluggish to light.

Case VII, No. 31. Infirmary. John Edwards, aged fifty-nine years, sailor. Complete right hemiplegia of four months' duration. Movement is present to some extent in arm and leg.

Sensations: Upper limb—Touch normal, pain hypo (slight), to heat normal, cold taken for heat.

Trunk—Touch normal, pain and temperature normal.

Lower limb—Touch normal, pain normal; to heat delay on foot, above normal; cold taken for heat from ankle up, normal on foot.

Face—Touch normal, pain and temperature normal.

Astereognosis absent. Position of paralyzed arm cannot be found with the normal limb. Special senses normal. Paresthesia absent. Hospital record shows analgesia and astereognosis. Patient says sensation improved when movement improved.

Case VIII, No. 29. Infirmary. Charles Bratton, aged sixty years, laborer. Complete right hemiplegia of a few days' duration. Movements improved, arm in better condition than leg.

Sensations: Upper limb—Touch normal, pain normal, to heat normal, to cold normal.

Trunk—Touch normal, pain normal and temperature normal.

Leg—Touch normal, pain hypalgesia, to heat hypoesthesia, to cold normal.

Face—Touch normal, pain hypalgesia, to heat normal, to cold normal.

Astereognosis absent. Position of paralyzed limbs recognized. Special senses normal. No paresthesia.

Case IX, No. 22. Infirmary. John McAnally, aged seventy years, laborer. Complete right hemiplegia of seven years' duration. (Leg only slightly involved.) Movements improved. Speech impaired.

Sensations: Upper limb—Touch normal, pain normal.

Thorax, lower limb and face normal to touch, pain and temperature.

Position of paralyzed arm could not be determined. Astereognosis absent. Special senses normal. No paresthesia.

Case X, No. 32. Infirmary. Adolph Schultz, aged thirty-eight years, laborer. Complete left hemiplegia of five months' duration. Movements are better in leg than in arm.

Sensations: Upper limb—Touch hypesthesia (slight), pain hypalgesia (slight), to heat and cold normal.

Trunk—Touch normal, pain hypalgesia, to heat and cold normal.

Lower limb—Touch normal, pain hypalgesia, temperature normal.

Face—Touch and pain normal, temperature normal.

Astereognosis absent. Position of paralyzed limbs not easily recognized. Special senses normal. No paresthesia.

Case XI, No. 2. Women's Ward. Jane Blair, aged eighty-two years. Complete right hemiplegia of three and one half years' duration. Movements in leg much better than in arm.

Sensations: Upper limb—Touch normal, pain hypalgesia on posterior aspect, normal on anterior; to heat hypesthesia on arm and forearm, normal on hand, to cold normal.

Trunk—Touch hypesthesia, pain normal, to heat and cold normal.

Lower limb—Touch normal, pain normal and temperature normal.

Face—Touch and pain normal, to heat normal, to cold hypesthesia.

Astereognosis is present only for consistency of objects. Special senses normal. No paresthesia.

Case XII, No. 4. Women's Ward. Mary Doyle, aged fifty-two years. Left hemiplegia (except face) of one year's duration. Movements much improved in leg, in arm no movements.

Sensations: Upper limb—Touch and pain normal; heat taken for cold from elbow up, normal below, except a few areas of hypesthesia, to cold normal.

Trunk—Touch normal, pain hypalgesia, heat taken for cold, to cold normal.

Lower limb—Touch normal, pain hypalgesia, to heat normal, to cold normal.

Astereognosis present only for size of small objects. Position easily recognized.

Case XIII, No. 24. Women's Ward. Annie Moore, aged forty-eight years. Complete left hemiplegia of four years' duration. Movements better in leg than in arm.

Sensations: Upper limb—Touch normal, pain hypalgesia and delay, to heat hypesthesia (marked), to cold almost anesthesia.

Trunk—Touch hypesthesia, pain hypalgesia, heat taken for cold, cold taken for heat.

Lower limb—Touch hypesthesia, pain hypalgesia, to heat normal, to cold hypesthesia.

Face—Touch hypesthesia, pain hypalgesia, to heat normal, to cold hypesthesia.

Astereognosis very marked for consistency, shape and size of objects. Position cannot be recognized. No paresthesia. Special senses normal.

Case XIV, No. 26. Women's Ward. Alice Facett, aged forty-nine years. Complete right hemiplegia, impairment of speech. Movements are present somewhat in leg, none in arm.

Sensations: Upper limb—Touch hypesthesia, pain analgesia, to heat hypesthesia (marked), to cold hypesthesia (marked).

Trunk—Touch anesthesia, pain analgesia, to heat normal, to cold hypesthesia.

Lower limb—Touch hypesthesia, pain analgesia (almost), to heat and cold anesthesia.

Face—Touch anesthesia, pain analgesia, to heat and cold hypesthesia.

Astereognosis is present. Position impossible to determine. No paresthesia. Special senses normal.

Case XV, No. 19. Women's Ward. Mary Ford, aged seventy-two years. Complete left hemiplegia of five months' duration. Movements improved considerably; more power in leg than in arm.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia.

Trunk—Touch normal, pain hypalgesia, to heat normal, to cold hypesthesia.

Lower limb—Touch normal, pain normal, to heat and cold hypesthesia.

Face—Touch normal, pain hypalgesia, to heat normal, to cold hypesthesia.

Astereognosis present. Position is not easily recognized. Special senses are normal. No paresthesia.

Case XVI. No. 18. Women's Ward. Rachel Brandon, aged forty years. Complete left hemiplegia of four years' duration. Movements improved more in leg than in arm.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia.

Trunk—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia (slight).

Lower limb—Touch and pain hypesthesia, to heat and cold hypesthesia (slight).

Face—Touch, temperature and pain hypesthesia (slight).

Astereognosis present. Position can easily be recognized. No paresthesia. Special senses normal.

Case XVII, No. 14. Women's Ward. Theresa Gallaguer, aged sixty-eight years. Complete right hemiplegia of one year's duration. Movements improved much in leg, but very little in upper limb.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia.

Trunk—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia (slight).

Lower limb—Touch normal, pain hypalgesia from foot up to knee, above normal; to heat and cold hypesthesia from foot to knee, above normal.

Face—Touch normal, pain hypalgesia, to heat hypesthesia, to cold normal.

Astereognosis present; recognizes shape and consistency, but not object. Position easily recognized. Special senses normal.

Case XVIII, No. 2. Women's Ward. Mary Griffin, aged eighty-five years. Right hemiplegia of twenty-three years' duration. Movements improved in leg, but arm contracted.

Sensations: Upper limb—Touch normal, pain normal, to heat and cold normal.

Trunk—Touch and pain normal, to heat and cold hypesthesia.

Lower limb—Touch, temperature and pain normal.

Astereognosis is present; recognizes size, but not shape, and cannot tell name of object. No paresthesia.

Case XIX, No. 20. Women's Ward. Catherine Boyle, aged fifty-five years. Complete right hemiplegia of one and one half years' duration. Movements improved; upper limb in better condition than lower.

Sensations: Upper limb—Touch normal, pain hypalgesia, heat taken for cold on inner surface, hypesthesia on external aspect, to cold hypesthesia (slight).

Trunk—Touch normal, pain hypalgesia (slight), to heat hypesthesia, to cold normal.

Lower limb—Touch normal, pain hypalgesia (slight), to heat and cold hypesthesia.

Face—Touch normal, pain hypalgesia (slight), to heat hypesthesia (slight), to cold normal.

Astereognosis present: does not recognize size of objects. Position is not easily recognized. Paresthesia present. Special senses normal.

Case XX, No. 26. Women's Ward. Mary Jesse, aged forty-six years. Complete right hemiplegia of eighteen months' duration. Movements are better in arm than in leg, which is contracted.

Sensations: Upper limb—Touch anesthesia, pain analgesia, to heat anesthesia, to cold anesthesia in hand, hypesthesia in forearm, returns to normal at the root.

Trunk—Touch anesthesia, pain analgesia, to heat anesthesia, to cold some areas anesthesia, some areas hypesthesia, in some areas taken for heat.

Lower limb—Touch anesthesia, pain analgesia, to heat and cold anesthesia, but returns to normal towards the hip.

Face—Touch hypesthesia, pain analgesia, to heat anesthesia, to cold hypesthesia.

Patient has pharyngeal anesthesia, color-fields for white and red contracted. Astereognosis is present. Position not well recognized. Special senses: right pupil large and sluggish to light, gray degeneration of both optic nerves, more in temporal halves. Paresthesia present.

Case XXI, No. 25. Women's Ward. Anna Williams, aged sixty-two years. Complete right hemiplegia of four months' duration. Movements are better in leg than in arm.

Sensations: Upper limb—Touch hypesthesia (marked), pain analgesia, to heat anesthesia on fingers, above hypesthesia; cold taken for heat.

Trunk—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia.

Lower limb—Touch hypesthesia, pain hypalgesia (slight), to heat hypesthesia (slight), to cold normal.

Astereognosis present: recognizes size with difficulty and cannot recognize objects. Position not easily recognized. Special senses: hearing impaired on paralyzed side. Paresthesia present.

Case XXII, No. 15. Women's Ward. Eva Bodge, aged twenty-six years. Complete left hemiplegia since the age of three months. Movements are better in leg, but fairly good in both.

Sensations: Upper limb—Touch normal, pain hypalgesia, to heat hypesthesia, to cold normal.

Trunk and lower limb—Touch, temperature and pain normal.

Face—Normal to three sensations.

Astereognosis present for recognizing objects. Position not easily recognized. Special senses normal. No paresthesia. Marked stigmata of hysteria present. Epileptic seizures since infancy (age three months).

Case XXIII, No. 11. Women's Ward. Mary Bowe, aged sixty-eight years. Complete right hemiplegia of three years' duration. Movements improved, better in leg than in arm.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat hypesthesia, to cold hypesthesia (slight).

Trunk—Touch and pain normal, to heat hypesthesia, to cold normal.

Lower limb—Touch, temperature and pain normal.

Face—Normal for three sensations.

Astereognosis present: cannot tell objects. Position not easily recognized. Special senses normal. No paresthesia.

Case XXIV, No. 15. Ward I. Richard Pryor, aged fifty-five years, laborer. Complete right hemiplegia (slight) of two months' duration. Arm in much better condition than leg.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia.

Trunk—Touch normal, pain hypalgesia, to heat and cold hypesthesia.

Lower limb—Touch normal, pain hypalgesia, to cold hypesthesia on foot and thigh, normal on leg, to heat hypesthesia.

Face—Touch normal, pain hypalgesia, to heat and cold hypesthesia.

Astereognosis present: does not recognize objects. Position is easily recognized. Special senses normal. No paresthesia.

Case XXV, No. 15. Ward II. Thomas Daly, aged fifty-nine years, engineer. Complete left hemiplegia of one year's duration. Movements better in leg than in arm; in both movements are fair.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat anesthesia, cold taken for heat.

Trunk—Touch hypesthesia, pain hypalgesia, to heat hypesthesia, cold taken for heat.

Lower limb—Touch hypesthesia, pain hypalgesia (marked), to heat anesthesia, cold taken for heat.

Face—Touch hypesthesia, pain hypalgesia, heat taken for cold, cold taken for heat.

Astereognosis present. Position easily recognized. Special senses normal. No paresthesia.

Case XXVI, No. 9. Ward I. Samuel Butters, aged forty-four years, dentist. Complete left hemiplegia of three years' duration. Movements better in arm than in leg; in both limbs movements are slight.

Sensations: Upper limb—Touch hyperesthesia, pain hypalgesia, to heat hypesthesia from wrist above, below normal, to cold hypesthesia from wrist above, normal below.

Trunk—Touch and pain hypesthesia, to heat normal, to cold hypesthesia (slight).

Lower limb—Touch normal, pain hypalgesia, to heat hypesthesia (slight), to cold hypesthesia.

Astereognosis present for shape of objects. Special senses are impaired: hearing is somewhat impaired. No paresthesia.

Case XXVII, No. 17. Ward I. Colin McPherson, aged sixty years. Complete left hemiplegia of six years' duration. Movements better in leg than in arm; fairly good in both.

Sensations: Upper limb—Touch hypesthesia, pain normal, to heat hypesthesia on forearm, normal above and below, to cold normal.

Trunk—Touch, temperature and pain normal.

Lower limb—Touch and pain normal, to heat and cold hypesthesia.

Face—Touch, temperature and pain normal.

Astereognosis present: cannot recognize objects. Position easily recognized. Special senses: slightly deaf on the paralyzed side. Paresthesia present.

Case XXVIII, No. 14. Ward II. James Egan, aged seventy-four years, baker. Complete left hemiplegia of four and a half years' duration. Movements very much improved in arm and leg, better in leg.

Sensations: Upper limb—Touch anesthesia, pain hypalgesia, to cold hypesthesia (slight), to heat anesthesia from wrist up, hypesthesia below.

Trunk—Touch anesthesia, pain hypalgesia, heat taken for cold, to cold hypesthesia.

Lower limb—Touch anesthesia, pain hypalgesia (marked), to heat anesthesia, to cold hypesthesia.

Face—Touch normal, pain hypalgesia, to heat anesthesia, to cold hypesthesia.

Astereognosis present: does not recognize objects and their consistency. Position not easily recognized. Paresthesia present. Special senses: hearing impaired.

Case XXIX, Ward V. Peter Kunsick, aged fifty-seven years, machinist. Complete left hemiplegia of two years' duration. Movements better in leg than arm.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat hypesthesia, to cold hyperesthesia.

Trunk—Touch hypesthesia, pain hypalgesia, to heat hypesthesia, to cold hyperesthesia.

Lower limb—Touch hypesthesia, pain hypalgesia, to heat hypesthesia, to cold hypesthesia, except on ankle, where it is taken for heat.

Face—Touch normal, pain hypalgesia, to heat hypesthesia, to cold hyperesthesia.

Astereognosis present for size and objects. Position not easily recognized. Special senses involved; hearing impaired. Pupil smaller than on other side. Paresthesia present.

Case XXX. Ward III. Isaac Jacobs, aged fifty-two years, porter. Complete left hemiplegia of eighteen months' duration. Movements better in leg than in arm.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat anesthesia, to cold hypesthesia.

Trunk—Touch normal, pain hypalgesia, to heat hypesthesia (slight), to cold normal.

Lower limb—Touch normal, pain hypalgesia, to heat normal, to cold hypesthesia.

Face—Touch and pain normal, to heat and cold hypesthesia (slight).

Astereognosis present. Position easily recognized. Special senses normal. No paresthesia.

Case XXXI, No. 7. Ward III. James Howard, aged sixty-two years. Complete left hemiplegia of two years' duration. Movements slightly improved in arm, but no movements in leg.

Sensations: Upper limb—Touch hypesthesia (slight), pain hypalgesia, to heat and cold hypesthesia.

Trunk—Touch hypesthesia, pain hypalgesia (marked), to heat hypesthesia (marked), cold taken for heat.

Lower limb—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia.

Face—Touch hypesthesia, pain hypalgesia (slight), to heat hypesthesia (marked), to cold hypesthesia.

Astereognosis present, position not easily recognized. Special senses: slight impairment of hearing. Paresthesia present.

Case XXXII, No. 24. Ward V. Jeremiah Boland, aged seventy-two years. Complete right hemiplegia of seven years' duration. Moves arm fairly well, the leg very little.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat and cold hypesthesia.

Trunk—Touch normal, pain hypalgesia, to heat and cold hypesthesia.

Lower limb—Touch normal, pain hypalgesia, to heat and cold hypesthesia.

Face—Touch normal, pain hypalgesia, to heat and cold hyperesthesia.

Astereognosis very slight. Position not easily recognized. Special senses: hearing impaired, right pupil larger than left. No paresthesia.

Case XXXIII, No. 17. Ward V. J. M. McGranan, aged fifty-nine years, carpenter. Complete left hemiplegia of seven years' duration. Movements are absent in arm and leg.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia (marked), to heat hypesthesia (marked), cold taken for heat.

Trunk—Touch hypesthesia (slight), pain hypalgesia (marked), to heat anesthesia, to cold hypesthesia (marked).

Lower limb—Touch hypesthesia, pain hypalgesia, heat taken for cold, to cold hypesthesia (marked).

Face—Touch, temperature and pain hypesthesia.

Astereognosis present. Position easily recognized. Special senses: hearing impaired. Left pupil smaller. Paresthesia present.

Case XXXIV. Hollon Boyd, aged forty-two years, Jefferson Hospital. Complete hemiplegia of eight years' duration. Movements in leg much better than in arm.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, to heat hypesthesia, cold taken for heat.

Trunk—Touch hypesthesia, pain hypalgesia, to heat hypesthesia in some areas, in others taken for cold; cold normal.

Lower limb—Touch and pain normal, to heat normal, cold taken for heat.

Face—Touch and pain normal, to heat normal, cold taken for heat.

Astereognosis complete. Position not easily recognized. Paresthesia present.

Case XXXV. Charles Sommers, aged sixty-four years, driver, Jefferson Hospital. Complete right hemiplegia of one and one half years' duration. Movements better in leg than in arm.

Sensations: Upper limb—Touch hypesthesia, pain hypalgesia, heat taken for cold, to cold hypesthesia.

Trunk—Touch hypesthesia, pain hypalgesia, heat taken for cold, to cold hypesthesia.

Lower limb—Touch anesthesia, pain analgesia, heat taken for cold on thigh, anesthesia on leg, to cold anesthesia.

Face—Touch, temperature and pain hypesthesia.

Astereognosis present. Position could not be determined. Paresthesia present.

The disturbances of sensations in these thirty-five cases in order of frequency are:

### TOUCH

Upper limb—Hypesthesia (19 cases), normal (11), anesthesia (4), hyperesthesia (1).

Lower limb—Normal (21), hypesthesia (10), anesthesia (5), hyperesthesia (1).

Trunk—Normal (18), hypesthesia (13), anesthesia (4), hyperesthesia (0).

Face—Normal (22), hypesthesia (8), anesthesia (1), hyperesthesia (0).

### PAIN

Upper limb—Hypalgesia (25), normal (6), analgesia (5), hyperalgesia (0).

Lower limb—Hypalgesia (21), normal (11), analgesia (2), hyperalgesia (1).

Trunk—Hypalgesia (24), normal (9), analgesia (3), hyperalgesia (0).

Face—Hypalgesia (15), normal (12), analgesia (4), hyperalgesia (0).

### TEMPERATURE

Upper limb, heat—Hypesthesia (20), normal (9), anesthesia (8), reversed (4), hyperesthesia (1).

Cold—Hypesthesia (17), normal (9), reversed (5), anesthesia (3), hyperesthesia (3).

Lower limb, heat—Hypesthesia (14), normal (12), anesthesia (8), reversed (3), hyperesthesia (0).

Cold—Hypesthesia (17), normal (13), reversed (4), anesthesia (3), hyperesthesia (1).

Trunk, heat—Hypesthesia (15), normal (11), reversed (5), anesthesia (3), hyperesthesia (0).

Cold—Hypesthesia (15), normal (13), reversed (4), hyperesthesia (3), anesthesia (1).

Face, heat—Normal (14), hypesthesia (12), reversed (2), anesthesia (2), hyperesthesia (1).

Cold—Hypesthesia (13), normal (11), hyperesthesia (3), reversed (2), anesthesia (0).

Astereognosis—Complete in 22 cases, partial in 7 cases.

Position of paralyzed arm—Abnormal in 17 cases, normal in 11 cases.

Paresthesiæ present in 12 cases.

Special senses involved in 11 cases.

The relative frequency of sensory disturbances in different parts of the body:

Upper limb—All three forms of sensations are affected in the majority of cases: for touch we find 24 cases, for pain 30 and for temperature 33 (H) and 28 (C). The hypo-sensations take the greatest number; the anesthesias come next and only a few cases present hyperesthesias. Among the thermic disturbances we find also a reversed condition in some cases, namely, heat is taken for cold and *vice versa.*

Lower limb—The disturbances are as follows: For touch 16 cases, for pain 24, for temperature 25 (H), 25 (C). The hypo-sensations take the largest number, the anesthesias come next, a few present hyperesthesias. As in the upper limb we also find a few cases with a reversed temperature sense.

Trunk—For touch we find disturbances in 17 cases, for pain in 27, for temperature 23 (H), 23 (C). Reversed temperature sense is found in a few cases.

Face—For touch the disturbances are in 9 cases, for pain in 19, for temperature in 17 (H), 18 (C), also reversed temperature sense is present in a few cases.

We therefore see that, without exception, in all the four portions of the body the pain sense suffers most. Next is temperature, and touch is the least affected. There is also no exception to the fact that whether in pain, touch or temperature, the hypo-sensation takes the largest number of cases, while anesthesias are met with in a comparatively small number, the hyperesthesias in extremely few cases.

It will be also noticed that the largest number of cases has the upper extremity affected with sensory disturbances: this is true for each of the three forms of sensations.

In the lower limb touch and pain are affected in a slightly smaller number of cases than on the trunk, except the temperature sense, which suffers more in the leg than on the trunk.

The face is the least affected.

This can be summarized as follows: Touch—Upper limb, trunk, lower limb, face. Pain—Upper limb, trunk, lower limb, face. Temperature—Upper limb, lower limb, trunk, face.

Verger states that the disturbed sensations have a tendency to become normal from the distal end of the limb towards the root. This assertion I found to be correct in a large number of my cases.

The stereognostic sense was disturbed in 29 cases out of 35, in 22 of which the loss was complete. The greater the involvement of the other sensations, the more marked was astereognosis, a fact which tends to favor the conception of the stereognostic sense as depending upon the integrity of the three cardinal sensations. The sense of posture for the upper limb was involved in but 17 cases out of the 35, a fact which leads to the idea that disturbances of other sensations do not necessarily involve the posture sense; this should be particularly emphasized, since the sensations of the upper limb were disturbed in the great majority of our cases.

Verger and others speak of paresthesia and disturbance of the special senses. As to the first I found it present only in 12 cases.

The special senses were involved only in 11 cases. Again the last two symptoms were present only in these cases, in which the other sensations suffered to a great extent.

Accepting the fact that sensory disturbances are present, it is interesting to see in what relationship they are to the degree of motor impairment. In 23 cases in which the impairment of motor power was less marked in the lower than in the upper limb, the majority (15 cases) presented less sensory disturbances; six cases, equal sensory disturbances, and only two more sensory disturbances than in the arm. The reverse is the case with the arm: out of eight cases, in which the motor power was better than in the lower limb, in five the sensory disturbances were more marked, in two they were less marked, and in one—equal to those of the leg.

Can we draw any conclusion in regard to a parallelism between motor and sensory disturbances? Taking into consideration the statement mentioned above, that the sensations are disturbed in a far larger number of cases in the upper limb than in any other portion of the body, and that in the majority of cases the motor power in the upper limb suffers more than in the lower limb, we are warranted to conclude in favor of a certain parallelism between motor and sensory disturbances. In favor of this view speaks also the fact that in those cases in which the motor paralysis was not marked the sensory disturbances were also less marked, and in those cases in which the motor paralysis was much pronounced the sensory disturbances were equally marked.

Is there any relation between the sensory disturbances and the date of the motor paralysis?

I found that in motor palsies of two years', and less, duration, the sensory disturbances are marked in largest number of cases, 13 out of 17. In cases of from two to eight years' duration we find eight in which sensory disturbances are marked, and in three slightly marked. Above this age the sensory disturbances are less marked. It is, therefore, important to note that as a rule the longer the date of motor palsy the less the sensory disturbances are marked.

The results obtained from this study appear to me to be very instructive. Without possessing the pathological data to confirm what my clinical investigations show, can we consider the sensory disturbances in all my cases merely as a coincidence, or as Legroux, Brun, and Mills suggest, that "circulatory disturbances and pressure on neighboring parts beyond those included in the paralyzing lesion account for the impairment of sensation"?

As to the last assertion it is hardly admissible that such a uniformity in the sensory disturbances in my cases should be due to that cause. A great number of cases were recorded by very careful observers in which the lesions were strictly limited to the Rolandic region and in which sensory disturbances were present. On the other hand we know of cases reported by equally careful observers, in which lesions were found in the areas supposed to be sensory while the disturbances were identical with those which were stated in lesions of the Rolandic region.

It is superfluous to go over all the discussions on the sensory localizations. They are too numerous and too well known to the partisans of both doctrines. I merely ask the question: What opinion can we form of the observations in which a lesion strictly limited to the motor area gave sensory disturbances and a lesion of the supposed sensory area gave place also to motor disturbances?

It is true that Dercum and Spiller's case may answer this question but the case is unique and stands isolated, and we have there besides a primary involvement of the *carrefour sensitif,* also one of the lenticular nucleus.

While I was studying these cases, I was surprised at the uniformity of the results, just as if the patients had entered into a mutual agreement to present sensory disturbances. I therefore asked myself, if the negative clinical results obtained by the partisans of the doctrine of separate localizations were really negative, for I found that it is not sufficient to content one's self with the statement that such and such a sensation is present. A persistent and repeated investigation will always (at least in my cases) elicit a more or less marked difference in the condition of the sensations on the paralyzed and non-paralyzed sides.

The writers, against whose views these thirty-five cases speak so loudly, claim that in a number of cases in which hemisensory disturbances were found to be present, they may have been due to hysteria. To avoid such a possible error, I intentionally excluded from my series cases with hysterical stigmata, for which symptoms I examined all the cases offered for my investigation.

However, I included one case (XXII) with distinct hysterical stigmata; here, in spite of the presence of this neurosis, the sensory disturbances are not hysterical in their nature, but of the same character as in the other cases.

A conclusion which forces itself upon me is that hemisensory disturbances, *probably*, always accompany a motor paralysis of cerebral origin no matter to what extent the latter is marked: paralytic or even only paretic symptoms will at the same time show sensory symptoms. The demonstration of this depends upon the method and persistence of the investigation.

If this study does not decide completely the problem of sensory and motor localizations for want of pathological data it may, nevertheless, serve as a guide to those who will have the opportunity to make pathological researches. It will stimulate them to examine very carefully the pathological findings. The clinical facts as described in this essay, cannot, and I hope, will not be ignored, for they are based on impartially and carefully collected observations.

Finally this work may serve its purpose by calling the pathologist's attention to the fact that he cannot be too careful in his deductions upon localizations.

I wish to express my indebtedness to Drs. Spiller, Potts and Dercum for their kind permission to use the material of their respective wards.

# REPORT OF A CASE OF FAMILIAL TREMOR OF THE HEAD.[1]

By John K. Mitchell, M.D.

The patient is twenty-two years of age, a student in one of the great Universities. He has had no illnesses beyond those of childhood. As a result of overwork in the laboratory last Winter, he became very nervous, readily excited and irritable, and in January of the present year first noticed the trouble of which he now complains, which has persisted steadily ever since.

He is a slight but well-built and muscular man, taking a good deal of exercise and without any evidences of impaired general health. It was not until after the general nervousness had lasted for some months that he observed that he had a slight lateral tremor of the head. The tremor is a very small one, moderately rapid and almost absolutely constant. It does not appear to be rendered worse by observation, it is not overcome by muscular effort although sometimes during muscular effort it ceases; it cannot be voluntarily stopped, and the effort to stop it does not alter its character. There are no evidences of involvement of the cranial nerves in any way. There are no points of tenderness in the cervical region. The head can be moved with entire freedom in every direction without any pain or spasm. He presents no sign of exophthalmic goiter. His reflexes are universally perfect, his general health is good, and the only thing that could be discovered calling for treatment was a myopic astigmatism very imperfectly corrected.

The point which leads me to report briefly this curious if not very important case is that the difficulty is a familial one. His mother had the same trouble, beginning at twenty-two years of age, and it has continued ever since. It has at times in her case been made worse by illnesses and then gone back to its previous standard on recovery from the acute illness. The patient's maternal grandfather developed a lateral tremor of the head at forty years of age and a maternal great-grandfather exhibited it at seventy, but no detail of the trouble in their cases can be had, though they are said to have had no other signs of paralysis agitans. It is possible, of course, that in these instances the two older patients may have been suffering with paralysis agitans, but it is unusual to see it begin in the head before it appears in other parts.

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[1] Read before the Philadelphia Neurological Society, November 25, 1902.

# Society Proceedings

## PHILADELPHIA NEUROLOGICAL SOCIETY.

November 25, 1902.

The President, Dr. John K. Mitchell, in the chair.

Drs. D. L. Edsall and R. S. Lavinson presented a case of senile chorea.

Dr. Alfred Gordon referred to the fact that the majority of patients with senile chorea present symptoms of dementia. Bishop, who has collected a number of these cases, states that sixty per cent. show symptoms of dementia.

With regard to treatment, he stated that in three cases of his own the use of antipyrin had been followed by improvement. Arsenic did not give beneficial results.

Dr. J. K. Mitchell referred to three cases which he had seen in different generations of the same family. The first case was that of a grandmother, seen twenty years ago; the second, that of a young woman just married, and the third a case of her child about two or three years old. They all presented typical Sydenham's chorea.

Dr. Alfred Gordon presented a woman with sensory and trophic disturbances of one limb, and also read a paper on traumatic syringomyelia.

Dr. F. X. Dercum said that the case was unique, and that in some of its physical features it resembled one presented by himself at the last meeting of the Society. In his case there were no symptoms of syringomyelia—simply a local swelling of the connective tissue resembling myxedema.

Dr. F. S. Pearce thought that the fact that the disturbance of sensation had extended to the face was rather against the diagnosis of syringomyelia. There must be something beyond the cord; and he could not agree that the changes were confined to the cord; therefore, it was very possible that trauma had to do with the etiology. The trophic changes were very interesting and he thought that their presence would hamper the determination of the exact sensory conditions present.

Dr. William G. Spiller said that he had seen sensory disturbance in the face in syringomyelia, and this disturbance does not necessarily indicate that the lesion is situated higher than the spinal cord or medulla oblongata. The spinal root of the fifth nerve extends down to about the second cervical segment, and this descending root is doubtless concerned with the sensation of the face.

Dr. W. G. Spiller read a paper on traumatic lesions of the spinal cord without fracture of the vertebræ, and showed by a case with necropsy that it is impossible to make a positive clinical diagnosis between central hematomyelia and traumatic myelitis.

Dr. F. S. Pearce said he had recently seen a case in consultation with Dr. Rodman—that of a woman who had fallen down stairs in somersault fashion, striking her head and falling about a dozen steps. She was immediately paralyzed in all four extremities. The X-ray failed to show any fracture of the vertebræ. The accident occurred last August. The patient is said to have had hysteria, and is now hysterical. She is in a condition of diplegia. She has atrophy of the thenar and hypothenar and interossei muscles, and of the muscles of the lower extremities with lessening of the reflexes of these limbs. The head is not involved. The sensory change has been hypesthesia, especially of the anterior aspect of both legs. She has incontinence and retention of urine, but is able to control the anal sphincter. This seems to be a case in which trauma has produced a myelitis, the prognosis being grave.

Dr. F. X. Dercum reported a case of trauma of the brain, followed by ataxia and focal epilepsy; marked improvement after operation.

Dr. William G. Spiller described a case seen by him last Summer, a man picked up by the side of a railroad who probably had been struck by a locomotive. He had convulsions confined to the left side of the body, and in these convulsions the head and eyes were turned very markedly to the left. This clinical observation supports the view that the center for conjugate deviation must be near the motor centers of the limbs, because if these centers were widely separated, we would not expect in a case like this, in which the convulsions were confined to one side of the body, to find conjugate deviation of the head and eyes.

Dr. Alfred Gordon remarked that the fact that focal epilepsy had been observed in cases where there was tumor of the frontal lobe, showed that the view that focal epilepsy was always due to lesion of the so-called motor area, was not absolutely correct.

Dr. J. K. Mitchell reported a case of familial tremor of the head.

# Periscope.

## DEUTSCHE ZEITSCHRIFT FUR NERVENHEILKUNDE

(Vol. 21, 1902. Heft. 5-6.)

21. Psychogenic Pseudo-Meningitis. Starck.
22. Periependymal Proliferation, Canal Formation, and Abnormal Developmental Processes in the Spinal Canal of Children. Rolly.
23. Criticism of Subcortical Sensory Aphasia. Strohmayer.
24. Clinical and Anatomico-Pathological Contribution upon Aphasia. Mingazzini.
25. Diffuse Sarcoma of the Pia Mater of the Whole Central Nervous System. Nonne.
26. A Contribution to the Knowledge of Disease of the Spinal Cord and Degeneration of the Antero-Lateral Tracts. Zahn.
27. Contribution to the Symptomatology and Diagnosis of Tumors of the Brain and of Chronic Hydrocephalus. Finkelnburg.

21.*Psychogenic Pseudo-Meningitis.*—Starck reports the following extraordinary case. A man thirty-one years of age was admitted to the hospital apparently suffering from some rigidity and pains in the neck. When examined it was found that he was suffering from lupus, and that he had had headache, vertigo, pain in the back, but no fever. Three days later he had vomiting and then a severe chill and some tinnitus. The stiffness in the neck continued, there was some sweating, persistent vomiting and loss of control of the bladder. After admission to the hospital the patient was somnolent, all the reflexes were greatly increased, and all forms of sensation were increased. A diagnosis of cerebrospinal meningitis probably tuberculous in nature, was made. The patient could not sleep, from time to time he had convulsions, and there was some gnashing of the teeth; finally distinct trismus developed. The urine obtained by catheter was normal. The pulse was abnormally slow and there was no fever. The symptoms, however, were somewhat suspicious. The convulsions were atypical and the patient's complaints of pain appeared excessive. An attempt was therefore made to hypnotize him which succeeded, and as a result of vigorous suggestion he recovered at once and was able to walk about and eat at the table with the other patients. His recovery was complete and he was discharged. A careful history was finally obtained. He had been an attendant in a hospital and subsequently had been a patient in a number of hospitals in which a diagnosis of meningitis had usually been made. His experience in Heidelburg under Starck had been the sixth as a patient and subsequently it was possible to trace his course in eight other hospitals. Finally in the next to last an operation was performed, the spinal canal opened, and the cerebrospinal fluid found to be under considerable pressure. He did not improve as a result of this until he was transferred to another hospital where he was treated firmly and finally was able to work. Altogether in his various experiences in hospitals, lumbar puncture was performed five times, and in only one case was there a doubtful elevation of pressure. Starck collects the reports of 17 cases from the literature which together with his own and another case of like nature he uses for the purpose of giving a sketch of the clinical history of the disease. There is usually an aura lasting several days or weeks, characterized by discomfort, headache, nausea, vomiting, loss of appetite and depression. Then follows a chill, delirium, fever, headache, stiffness of the neck and back, tenderness over the spinal column and cramp-like twitching of the muscles. Often there is strabismus, narrow pupils, slow pulse, and various paralytic and vasomotor disturbances. Often the body is retracted, and there is persistent vomiting and obstinate constipation. The delirium increases and finally passes into coma, then suddenly the patient has a prolonged period of sleep and awakes entirely well. Sometimes, however, the symptoms diminish gradually. The disease occurs much more frequently in women, 17 out of 19, and in early adult life, 2 cases having occurred in childhood, and 2 after forty-five years of age. There are often symptoms of nervous heredity, and occasionally of tuberculous antecedents. The diagnosis can often be suspected when hysterical stigmata are present. The difficulty most frequently occurs in connection with tuberculous meningitis, of course, although occasionally pseudomeningitis and traumatic tetanus may be suspected. Starck prefers the name "psychogenic meningitis" in order to distinguish it from the condition called pseudomeningitis by German writers, which occurs particularly in infectious fevers.

22. *Abnormal Developmental Spinal Canal.*—Rolly examined the spinal cords of two children suffering from so-called general congenital spasticity of the muscles, and found certain peculiar changes in the cord characterized by diffuse proliferation of the neuroglia tissue and some leptomeningitis. In addition to this there was in the first case apparently a doubling of the central canal with proliferation of the neuroglia cells, particularly in the posterior one. From time to time horizontal canals were found uniting these two. Carefully studied serial sections seemed to show that the cells surrounding the central canals which in all respects resembled glia cells, were derived from those of the ependyma. The size of the canal varied greatly: at times it was obliterated, at others quite large. In the second case of the same condition practically similar changes were found, particularly the alternation of a dilated with an obliterated canal. He therefore carefully studied the spinal cords of several other children, and in one of these found some proliferation of the neuroglia in the central canal. This child was an idiot. There was a general tremor when it sat up, and its movements indicated some spasm of the muscles. In this case there was a variable number of canals at different levels of the spinal cord. It is not impossible that the changes in this case may bear some relation to those found ordinarily in syringomyelia. He does not attempt to determine whether there is any relation between them and syphilis.

23. *Subcortical Sensory Aphasia.*—Strohmayer reports some cases of subcortical sensory aphasia. The first, a man of thirty-six years, who had had luetic infection eight years before, six years after this noticed loss of memory and rapid intellectual fatigue. He then had an epileptic attack followed by a second attack with transient paralysis of speech and pain in the right ear. After a third attack he was maniacal but energetic specific treatment apparently prevented any other attacks, although the pain in the right ear and the weakness of memory continued. When examined there were found several syphilitic manifestations, and it was noted that the patient heard badly and that he did not understand spoken words although he could hear faint sounds easily. Written speech he understood very well. The patient gradually grew worse, had athetoid movements, and finally died. A diagnosis of atypical dementia paralytica with focal symptoms of subcortical sensory aphasia in the sense of Wernicke-Lichtheim, was made. At the autopsy there was found an area of softening in the median anterior third of the left thalamus opticus, and an old extravasation of blood, in the right temporo-sphenoidal lobe between the second and third convolutions, and in both temporo-sphenoidal lobes some change in the appearance of the cortex which was found to be due to a fibrous leptomeningitis. Therefore, the chief change was a diffuse lesion of the cortex and not a subcortical focal lesion. Strohmayer has collected 12 cases of subcortical

aphasia which he tabulates and shows that various pathological lesions can cause the symptoms.

He reports a second case of his own, a woman of fifty-three years, who developed pain in the neck and back of the head, scotomata before the eyes, vomiting, loss of power to walk, vertigo, and peculiar weakness of memory, consisting of difficulty in recalling names. A diagnosis of cerebral tumor in the region of the anterior portion of the corpus callosum was made. At the autopsy a tumor was found about 20 mm. posterior to the end of the corpus callosum in the median line, about the size of a cherry. Tumors were also found in both cerebellar hemispheres, in the second right occipital lobe, in the right temporo-sphenoidal lobe, in the neighborhood of the second convolution, and an area of softening in the left temporo-sphenoidal lobe. These were all probably secondary to a carcinomatous neoplasm of the pleura. In this case subcortical lesion had not given rise to subcortical aphasia, and he therefore believes that we should discard this name entirely and restrict ourselves to the clinical term "word-blindness."

24. *Aphasia.*—Mingazzini reports the following case of aphasia: A woman, sixty-seven years of age who had been an alcoholic, at the age of sixty-three had had a fall injuring her head, following which there was headache. Then she began to have paraphasia, loss of the ability to understand questions, inability to dress herself, and confusion of the various implements that she used, such as knives and forks. Finally she had convulsions resembling epilepsy. The patient finally became unable to make more than unintelligible sounds and died. There was atrophy of the entire brain, the convolutions were smaller and Nissl's method showed changes in the pyramidal cells. The case illustrates the fact that progressive atrophy of the brain may lead to typical aphasic disturbances.

25. *Sarcoma of Pia Mater.*—Nonne reports the case of a girl sixteen years of age who had an attack characterized by a feeling of foreign body in the larynx and then pain radiating over various parts of the body. There were clonic movements in the extremities, but nevertheless a diagnosis of hysteria was made. Five months later the patient was readmitted with severe pains in the neck, back and lumbar region. Three weeks previously she had suddenly become blind. There was cachexia, and hallucinations of hearing and sight, but the patient was apathetic and weak-minded. She improved somewhat, the pupils showed extraordinary changes, alternating between mydriasis and myosis, one changing at a time and showing great irregularity of ordinary pupillary reactions. The patellar reflexes were lost. Later there was paralysis of both oculomotor nerves and there was some doubt as to whether there was atrophy of the optic nerves or not. Lumbar puncture showed an increased pressure. From time to time the patient could see and hear and then would again become blind and deaf. She finally died. A diagnosis of cerebral tumor in the region of the corpora quadrigemina with metastasis to the spinal column was made. At the autopsy an absolutely negative macroscopical brain and spinal cord were found. Microscopically, however, it was found that the whole pia was filled with a proliferation of new cells. This was of the character of sarcoma. The central nervous system appeared to be normal. Nonne discusses similar cases that have been reported, and believes that the extraordinary variation in the severity of the symptoms is due to the peculiar way in which the tumor mass affects the blood vessels. Although young persons are most frequently affected, cases have been reported occurring in individuals up to sixty years of age.

26. *Degeneration of Antero-lateral Tracts.*—Zahn reports the case of a girl twenty-six years of age, who at the age of sixteen had had difficulty

in speech and uncertain gait. Her father had died after having had the same disease for twenty-four years. When examined it was found that the muscles were spastic, there was no degeneration, the reflexes were greatly increased, ankle-clonus was present, muscular power was not diminished, and there were no defects in the organs. Sensation was apparently normal. The disturbance of speech was due to the involuntary movements of the muscles of articulation. The patient was under observation for some time, and her gait was improved by practice. She finally died as a result of septic peritonitis. At the autopsy the pia mater of the spinal cord was found irregularly thickened, and there was degeneration of the pyramidal tracts, although the intensity of the degeneration varied at different levels. There was slight degeneration of the posterior columns in the upper portion. Zahn does not believe that this irregular degeneration of the white substance of the cord is to be looked upon as secondary. The best explanation seems to be the assumption that the process is a sort of hereditary degeneration, and it does not appear likely that an accurate diagnosis of the condition could have been made during life.

27. *Tumors of Brain and Hydrocephalus.*—Finkelnburg has analyzed 67 cases of brain tumor occurring in the medical clinic at Bonn. He tabulates the cases according to the anatomical situation of the tumors. There were 22 cases of tumor of the cerebellum, 13 of which were confirmed by autopsy. In three cases operations were performed, one of which resulted successfully. In four cases confirmed by autopsy choked disk was absent. In three cases the patients had cardiac arhythmia and disturbance of respiration when the head was moved. There was considerable difference in the behavior of the reflexes. Some of the cases were exceedingly difficult to diagnose. He mentions one case, a child in the fourth year of life with difficulty in walking, enlargement of the head, choked disk, disturbance of sensation in the area of the right trigeminal nerve, and paresis of the lower facial. There was increased pressure in the spinal cord, and at the autopsy a sarcoma of the base of the right hemisphere was found with internal hydrocephalus. In another case the patient had a cerebellar gait, pain in the back of the head, weakness and vertigo. There was also nystagmus, increase in the tendon reflexes, Romberg's symptom, but no choked disk. Four years later the patient presented typical picture of multiple sclerosis. There were 27 cases of cerebral tumors sufficiently carefully studied for statistical purposes. The reflexes showed marked variations. Eight cases were subjected to operation five times merely to relieve the symptoms, and in two of these considerable improvement occurred. In three other cases the tumors were found at the operations. In two of these the patients died, and in the third the tumor was so large that only a part could be removed. Some of the cases were exceedingly interesting. In one commencing in the fifteenth year there was increased thirst, polyuria, gradual loss of sight, development of staggering gait and death, after 4½ years. At the autopsy sarcomatous proliferation was found in both ventricles. In another case the patient had nervousness, headache, vertigo, choked disks, increased tendon reflexes, cerebellar ataxia, but no focal symptoms. A round cell sarcoma was found on the floor of the fourth ventricle. In still another case the patient had headache, vertigo, staggering gait, vomiting, weakness in the left facial nerve, choked disks, alternating hemiplegia, progressive apathy, temporary improvement and finally death. A glioma was found in the third ventricle involving the right crus and tegmental region. Some interesting observations of tumors and other disease conditions of the base of the brain are given. In one case hemicrania had existed for 20 years, then gradual loss of sight in the left eye took place, loss of muscular control in the right eye, right hemiplegia and disturbance of speech. A diagnosis of tumor of the sella turcica was made, but an autopsy was not obtained. In another case multiple spindle-celled sarcomata were found,

but during the clinical course the patient had improved remarkably upon a mixed treatment. The histories of several other cases of multiple tumors are also given, also of several cases of chronic hydrocephalus simulating brain tumor, and Finkelnburg summarizes the symptoms of this condition as follows; Chronic development without distinct initial symptoms of meningitis; very slow course with marked remissions; at first symptoms of intracranial pressure, which may disappear entirely; or leave atrophy of the optic nerves only; absence of symptoms of focal disease; early occurrence of vision and weakening of the patellar reflexes.

J. Sailer (Philadelphia).

### NEUROLOGISCHES CENTRALBLATT

(Vol. 21, 1902, No. 20. Oct. 16.)

1. A Case of Arsenical Paralysis. J. Kron.
2. A Case of Katatonia following the First Menstrual Period. H. Mucha.
3. Paradoxical Pupillary Contraction and an Observation of Contraction of the Pupil by Shading the Eyes. J. Piltz.

1. *Arsenical Paralysis.*—Report of a case of neuro-arsenical neuritis following the ingestion of four grains of arsenic during twenty hours.

2. *Katatonia.*—Report of a case of typical katatonia during the first menstrual period. Mucha calls attention to the early age of his patient, fifteen years.

3. *Continued article.*

(Vol. 21, 1902, No. 21. Nov. 1.)

1. Psychology of Motor Apraxia. A. Pick.
2. The So-called "Myotonia" Slowness during Convergence of Pupils with Loss of Reflex to Light. Nonne.
3. Contribution to the Localization of Cerebral Hemianesthesia. Karl Schaffer.
4. Paradoxical Pupillary Contraction and an Observation of Contraction of the Pupil by Shading the Eyes. J. Piltz.

1. *Motor Apraxia.*—Report of a case of what Pick considers a pure motor apraxia. The attacks of inability to find the words he wanted to use, were intermittent in character, occurring every two weeks and lasted several days. The only motor phenomenon was a spasmodic contraction of the masseter.

2. *Myotonic Slowness.*—Nonne adds a case of myotonic contraction of the pupil to those already discussed in the *Centralblatt.* This case occurred in a patient with diabetes and anchylosis of the spine and the pupillary phenomenon was unilateral. The time reaction for dilation of the pupil after contraction was five minutes. Another case in an alcoholic was also mentioned.

3. *Cerebral Hemianesthesia.*—Schaffer gives the results of a careful examination of the brain of a boy of eighteen years who died from heart failure. He had for some time motor and sensory paralysis localized to one side of the body. The cerebral lesion affected the anterior and posterior central gyri and extended deep into the cerebral substance. The posterior limb of the internal capsule was affected only in its anterior third. The view of Dejerine and Long are carefully considered in connection with the findings of this case.

4. *To be continued.*

(Vol. 21, 1902, No. 22. Nov. 16.)

1. The Difference in Prognosis between Nerve Paralysis and Nerve Trunk Paralysis. L. Bruns.

2. Sluggishness in Accommodation and Convergence, or Myotonic Pupillary Movement? J. STRASBURGER.
3. Paradoxical Pupillary Reaction and an Observation on Contraction of the Pupil by Shading the Eyes. J. PILTZ.

1. *Nerve Plexus and Nerve Trunk Paralysis.*—Bruns gives a critical analysis of 70 cases, 15 of which were plexus paralysis. He discusses the cause, intensity, and the time of the lesion as essential factors in making the prognosis. In cases of plexus paralysis when an involvement of the roots is suspected, or as in some cases in which the spinal cord itself is similarly involved, the prognosis is in proportion unfavorable. He thinks that such lesions of the nerve roots occur much more frequently in plexus lesions than has heretofore been accepted. He therefore arrives at the conclusion that so far as prognosis is concerned, plexus paralysis should take a middle position between the favorable peripheral nerve palsies and the very unfavorable spinal palsies.

2. *Myotonic Sluggishness.*—A controversial article with Sänger, as to the propriety and grounds for calling the sluggish contraction noted by these two observers, "Myotonic Contraction."

3. *Pupillary Paradoxical Phenomenon.*—Report of a case of true paradoxical pupillary phenomenon. After a careful review of the literature and of the different varieties of irregular pupillary action to light and convergence, Piltz concludes that true paradoxical light reaction is an exceedingly rare symptom, which up to the present time has been met with only in organic disease of the central nervous system.

(Vol. 21, 1902, No. 23. Dec. 1.)

1. Is there an Autogenetic Regeneration of Nerve Fibers? A Contribution to the Neurone Theory. E. MUNZER.
2. Observations on Winter Therapy in Mountain Climates. B. LAQUER.

1. *Autogenetic Regeneration.*—Munzer after a careful study of the work of Bethe on the endogenous regeneration of nerve fibers in rabbits, supports his conclusions in so far as they pertain to the presence of newformed nerve fibers in the distal stump of a cut nerve, but holds that the production of these new fibers from the cut fibers themselves is not proven. He finds that at the proximal end of the distal stump a nodule forms, but that this node is in connection with the nerves of the neighboring tissues; that new nerves forming in this node are independent of "centralization" is therefore not proven.

2. *Winter Therapy.*—A paper on the indications and contraindications to high altitude treatment for functional nervous diseases.

D. J. McCARTHY (Philadelphia).

### PSYCHIATRISCH-NEUROLOGISCHE WOCHENSCHRIFT

(Vol. 4, 1902, No. 32.)

1. Concerning the Bias of Hospital Physicians as Medical-legal Experts. DR. PFAUSLER, Director in Valduna.

This article of Dr. Pfausler is a retort to a criticism by some members of the legal profession of an article of his appearing in No. 7 of the present volume of this journal, in which he had asserted the fitness of the hospital physician for this work over that of the general practitioners summoned as experts by the Court. He lays special stress upon the protection afforded to society when the insane criminal is committed to a hospital upon the examination of the hospital physician and compelled to remain there until discharged at his discretion, rather than being sent to prison after an examination by the so-called experts for a definite term and then turned loose in the community while still insane. In substantiating his position, Professor Kraepelin is quoted extensively.

(Vol. 4, 1902, Nos. 33 and 34.)

1. Was Mohammed an Epileptic? **Dr. med. M. L. Moharrem Bey.**

In this interesting article the author reopens this discussion, and from a purely medical standpoint reviews the biographical literature and traditions concerning the family history, the early life, and especially the first revelation, and following this experience the partially unconscious states into which the prophet so frequently lapsed when receiving other revelations; and concludes that there is no basis for the assumption that Mohammed was an epileptic. He calls attention to the character of the so-called attacks when he would lie down upon the floor and would be covered with a garment and have a pillow placed under his head by those near him, and distinguishes them from the genuine epileptic attacks. These attacks were not followed by stupor, but the prophet would arouse himself quickly and begin at once to relate his divine revelation. Furthermore, the character of his intelligence which was maintained until his death at sixty-three of an acute febrile disease rules out the possibility of his having suffered in any degree from epileptic dementia.

(Vol. 4, 1902, No. 34.)

1. One-sided Delirium. **Professor E. Bleuler.**

This singular condition occurred during a paralytic attack of a paretic of two years' standing and fifty-one years of age. The course of the disease up to the time of the occurrence of this attack had been characterized by several paretic seizures. In this attack which was of twelve hours' duration, there seemed to be a dissociation of the movements of the right and left upper extremities. When first seen in the morning the right hand and arm were in constant motion, sometimes purposeful, at other times purposeless. What the right hand disordered, the left hand put to rights, and at times the left attempted to restrain the right. This continued throughout the morning. At noon, sensation on the left side of the body was found to be unimpaired, and occasionally tests for the same elicited defensive movements from the right side; while tests on the right side simply intensified its movements. Both legs from the onset presented convulsive movements. Strong stimulation of the right side produced energetic convulsive, purposeful movements, as if the right side was struggling against a phantom, in which the left did not participate. Two hours later the diversion of the two sides had become even more marked. At this time an attempt to place the patient on his feet caused incoördinate movements of both lower limbs. In the evening the two hands acted more in unison for short periods. Later, suddenly an attack occurred in which the right hand made gestures as if trying to snatch up and toss away rats, and as if battling with a phantom man to whom the patient spoke threateningly, while the left hand gesticulated as if trying to defend the head from a swarm of bees. After getting on his feet, each leg struggled to walk towards its own side, but soon the right prevailed and the patient went about in a circle. When returned to bed the movements of the right side continued; the left had already subsided. This continued for about an hour when it increased greatly and was combined with powerful movements of the leg which required the administration of chloral. The movements were suspended during the sleep which followed. During the entire day the patient was able to comprehend questions. Spontaneous speech was "delirious." He remained quiet when the left hand was held but restraint of the right caused irritability. The eyes and head were drawn to the right. The patient had no memory of the attack the following day. Increased activity of the right side continued for two days.

His conclusion is that each hemisphere of the brain seems to be able to apprehend impressions of the outside world independently, and can carry out complicated movements corresponding with these impressions, and cites in support of this view, cases in which one-half of the brain has been sufficient to maintain a psychical personality.

Ross Defendorf (Middletown).

### JOURNAL OF MENTAL SCIENCE

(Vol. 48, 1902. October.)

1. Presidential Address Medico-Psychological Association. J. Wigglesworth.
2. A Statistical Contribution to the Pathology of Insanity. T. Duncan Greenlees.
3. Importance of Stimulus in Repair and Decay of the Nervous System. F. W. Mott.
4. Some Remarks on the Surgical Treatment of Insanity. Damer Harrisson.
5. Treatment of Incipient and Transient Mental States in General Hospitals. T. S. Clouston.
6. Medico-Psychological Statistics. C. Hubert Bond.
7. Observations on the Neuroglia Cell and its Processes. R. R. Leeper.
8. Pathogenesis of Diabetic Insanity. W. R. Dawson.
9. Care of Idiots and Imbeciles. J. H. Sproat.
10. Mental Symptoms and Bodily Disease. Nathan Raw.

1. *Medico-Psychological Society.*—This is the presidential address delivered at the sixty-first annual meeting. The general topic of the substances in the germ cell involved in the hereditary transmission of insanity is first discussed by the author, this leading up to an analysis of the family histories of some 3,445 insane patients admitted into the Rainhill Asylum during the past twelve years. Out of this entire number definite family history of hereditary taint was found in 28 per cent. Paternal and maternal influences were about equally important, in 350 patients, the fathers were insane in 185 instances and the mothers 165. On the question of parentage and sex liability he finds that female children are more numerously affected independent of the sex of the affected parent, a slight increase is noted for insane mothers transmitting insanity to daughters than to sons. On the general biological question of the transmission of acquired characteristics the author then makes some interesting observations. For juvenile general paresis the author believes that it owes its origin to antecedent parental syphilis and that the parasyphilitic influence has been exerted in the germ cells to modify their nutrition and thus lead to degenerations in the offspring. Certain forms of general paresis in the adult may have a like origin. The address is a scholarly one and contributes much to the discussion of this important problem.

2. *Pathology of Insanity.*—Dr. Greenlees of South Africa offers a statistical contribution on the condition of the various organs of those dying of insanity. It is too detailed to permit abstracting.

3. *Stimulus in Repair and Decay of the Nervous System.*—Dr. Mott first discusses the nature of stimulus from the physical viewpoint, then takes up the question on its psychological side and later considers the anatomical and physiological substrata of nervous energy. Nerve fibers, he says, have been shown to be incapable of fatigue and he makes some interesting observations on the functions of the myelin sheath which he maintains has other functions than that of serving as an insulator. Impulses transmitted by non-medullated fibers of visceral and vascular structures are of low intensity compared with the medullated fiber of somatic structures. The chemical constituents of myelin are found in the cerebrospinal fluid. The development of the myelin sheath is correlated with the ratio of incoming stimuli, those parts first stimulated being the first myelinated, and other experiments prove that stimulation causes the formation of mye-

lin. The reverse is true also and the absence of an inflow of nerve stimuli causes an atrophy of the protagon of the the myelin sheath. Regeneration seems to be dependent on stimulation. He then discusses the effects of excessive stimulus showing that katabolism is the normal concomitant of the passage of nerve stimuli and if the neurones are in a low state of nutritional equilibrium the processes of disintegration are in excess of those of integration. The vulnerable part of the neurone is that furthest away from the center and in poison states it is usually the outlying collaterals that are first affected. Many poisons thus produce a loss of durability, an abiotrophy and therefore a nutritional deficiency which interferes with the balance of repair to waste.

4. *Surgical Treatment of Insanity.*—The author reports three cases of traumatic insanity with recovery following operation.

5. *Treatment of Insane in General Hospitals.*—Dr. Clouston opens a discussion on this point saying that at the present time there are no facilities for treating the acute insane as other acute diseases are treated in general hospitals and that there exists a great need for better provisional care of the acute insane than now exists in many places in England. Asylum extension will not fill the need and he makes a strong plea for the development of psychopathic wards in general hospitals. The ideal system should be fourfold—(1) A mental ward in the hospital for incipient, transient, and suitable cases; (2) a reception hospital for certifiable cases of an acute character situated near a large town with plenty of nurses and plenty of medical attention; (3) an ordinary asylum for cases that run on from month to month; (4) a boarding out system for the really quiet and manageable cases who could be restored in a modified degree to family life.

6. *Medico-psychological Statistics.*—A series of tables for the recording of symptoms in order to bring out greater uniformity. Too detailed for abstracting.

7. *The Neuroglia Cell and Its Processes.*—Dr. R. Leeper contributes a few general suggestions on the possible functions of the neuroglia cells.

8. *Pathogenesis of Diabetic Insanity.*—A short note of uncertain foundations on this subject.

9. *Care of Idiots and Imbeciles.*—A short note on the custodial care of this class of defectives.

10. *Mental Symptoms and Bodily Disease.*—A criticism of existing legislation on the care of certain types of insanity—delirium tremens in particular. Of local interest solely.

JELLIFFE.

### AMERICAN JOURNAL OF INSANITY

(Vol. 59, 1902, No. 1. October.)

1. The Criteria of Insanity and the Problems of Psychiatry. ABBOT.
2. On the Typhoid Psychoses. FARRER.
3. Some Points in the Diagnosis of Dementia Præcox. DUNTON.
4. A Case of Huntington's Chorea with Autopsy. RUSK.
5. On Some Terminal Diseases in Melancholia. MEYER.
6. Hydriatic Procedures as an Adjunct in the Treatment of Insanity. DENT.
7. Medico-Legal Phases of the Vermont Observation Law for Criminal Insane. BERRY.
8. The Possible Influence of Rational Conversation on the Insane. BUCKLEY.

1. *The Criteria of Insanity and the Problems of Psychiatry.*—The definitions by Krafft-Ebing, Maudsley and Spitzka are quoted and their incompleteness shown, as they leave out of consideration largely the

condition of environment, or even entirely, which is the cardinal factor in judging a person's actions, whether sane or insane, then the individual's understanding or conception of his environment in all its elements, and whether his failure in this respect is due to ignorance or error, and the latter carries with it the motive or purpose of his action; then again whether the individual acts from choice or was unable to do or think differently. From these data insanity is defined as a morbid condition of the mind, which renders it impossible for the conscious individual to think, feel or act in relation to his environment, in accordance with the standards of his bringing up. Thus ascertaining what the essence of insanity is, its form is the next problem to be considered, and consists in ascertaining the ways in which the insane person is unable to think, feel and act in relation to his environment, in other words observation and description of his thoughts, feelings and actions, where detail is an essential, therefore not only is the individual's environment to be noted, but his conception of it, his affective reaction to it, and his acts. His delusions are to be recorded in his own words, not our judgment of them, and he is to be drawn out as to the ideas he entertains, whether oriented as to time, place and persons, of his perception of his duties and obligations to others, *etc.* Further, his memory as well as his capacity to receive, retain and recall impressions, and the character of the ideas associated in his mind are to be ascertained. His affect and his own expression of his feelings are to be noted. The motive of actions must be inquired into. Then as to the causes, whether in the environment, such as heredity and conditions causing anxiety, overwork or excesses, and in the individual himself. Various physical conditions induce abnormal mental states, and therefore these must be investigated, further various examinations of the blood, urine, excreta, *etc.*, made, as well as of the physiological chemical processes, and psychological investigations carried out. Clinical histories of such a character enhance the value of post-mortem data many fold. Nevertheless equipped laboratories are not essential for the careful study of psychiatric symptoms, for they are mental phenomena.

2. *On the Typhoid Psychoses.*—The author's résumé gives the essential data of this paper and is therefore quoted *verbatim:* "Typhoid fever attacking a sane person may leave him free from psychic symptoms or give rise to all gradations of mental disease; the severity of the symptoms does not necessarily stand in relation to the height of the fever or the profoundness of the infection; persons of psychopathic heredity are more prone to alienation, especially initial delirium, than those not thus burdened; a protracted exhausting fever predisposes to an asthenic psychosis; a second and third attack of typhoid is more likely to present mental troubles than the first. Concerning the special disease types, it is shown that initial delirium is the rarest form, exhibits the most rapid course and the worst prognosis, over 50 per cent. ending fatally; that it is essentially the expression of severe intoxication, the Nissl findings confirming this view; that it often causes errors in diagnosis; that any case of mental derangement with fever justifies the suspicion of typhoid. It is seen that the febrile psychoses are of greatest frequency and afford the best outlook; that 25 per cent., however, persists for varying lengths of time into and after convalescence; that they are especially due to the elevation of temperature and its consequences; that the asthenic psychoses (excepting conceptions delirantes isolees, and cases of typhoid collapse delirium), present long weary courses and a doubtful outlook, with evidences of serious cerebral changes; that they develop upon a basis of exhaustion, anemia and malnutrition; further, that a post-typhoid predisposition (irritable weakness) exists, upon which may develop late psychoses, with a dubious prognosis. As a result of the metabolic revolution accompanying severe

infections, it is seen that an attack of typhoid has been followed in many instances of so-called recoverable psychoses, by temporary improvement or recovery. Finally, that neither in its clinical nor anatomical picture is the typhoid psychosis distinctive; that the elements of intoxication, infection, temperature, exhaustion, anemia, of whatever origin, may produce similar or indistinguishable appearances; and that the determining factor of susceptibility to mental disorder, as well as often course and event, may be expressed in a word as the mental reaction-coefficient of the individual together with its physical basis."

3. *Some Points in the Diagnosis of Dementia Praecox.*—A mechanical irritability of the facial nerve has often been found to exist in a very pronounced manner and to be of diagnostic value when associated with mental aberration. It is obtained as follows: when the patient's cheek is tapped lightly with a percussion hammer just in front of the ear, being careful to exclude any visual impulse, a noticeable movement of the orbicularis palpebrarum occurs, varying in intensity from a marked contraction of the whole muscle, combined with that of other muscles of the face, to a slight tremor of the fibers of the inferior portion of the orbicularis. It is not present in all cases, but most pronounced in the later stages. The tendon phenomena are exaggerated in every case, superficial reflexes also increased. Sudden impulses are an important feature, occurring without any warning, the patient unable to give any reason for them and often denies them. Stupor or confusion is usually increased after the act. In the paranoid state delusional explanation of it may be given. The slow psychical reaction is an important diagnostic sign. It is difficult and often impossible to differentiate negativism from delusional resistance, therefore it is of doubtful diagnostic value.

4. *A Case of Huntington's Chorea with Autopsy.*—The patient, a clergyman, fifty-six years of age on admission, died with Huntington's chorea at the age of sixty. The results of the autopsy are summarized by the author as follows: "The essential lesion consists in the diminution in size of nervous elements generally, an increase in pigment content of the nerve cells, especially in those of the cerebellum; an overgrowth of neuroglia tissue—the relation of which to the nervous elements seems to be passive and possibly accounted for by the so-called 'tissue tension'—a shrinkage of the cells in the dorsal root ganglia with the analogous proliferation of the endothelial cells of their capsules, a pigmentary degeneration of the neuroglia, and a degeneration of the white matter matter about the periphery of the cord."

5. *On Some Terminal Disease in Melancholia.*—From the autopsies of 36 cases of melancholia the immediate causes of death were found to be phthisis in two cases, lobar pneumonia in four, bronchopneumonia in fourteen, pulmonary infarcts in six, sepsis (from cystitis, pyonephrosis and parotitis and twice perirectal abscesses), in four cases, enteritis in three, suffocation in one and suicide in one. Phthisis, lobar pneumonia, diphtheria and suicide are eliminated. The fourteen cases of bronchopneumonia were accounted for in one by fracture of several ribs, in seven accompanied a "central neuritis," in the remaining six devility, difficulty in swallowing and forced feeding. As this disease is the result of ingestion of foreign material, the danger from forcing feeble patients to swallow food is obvious. Certain dangers follow rectal feeding, two fatal cases having resulted from rectal ulceration and its sequela, further the rectum has been perforated, once by overdistension and bursting at the sigmoid flexure, in another by perforation by the rectal tube. Ulceration of the rectum often leads to cystitis. In pulmonary infarct patients died suddenly, even after all precautions as to quiet were maintained, and there are no means to prevent this termination. The case of suffocation occurred in a weakened patient from syncope from undue

exertion. The "insane ear" has of late become rare, and the better class of attendants is the reason, and many cases of internal hemorrhagic pachymeningitis are due to practically the same cause; therefore falls, bumps and bruises are to be guarded against in those patients whose tissues are degenerated. A debilitating diarrhea often attends cases of "central neuritis" or "systemic parenchymatous degenerations" mainly indicating the central nervous system. In depressive conditions patients often become feeble, take to their beds with variable attacks of diarrhea and often attended with a peculiar rigidity of nearly all the muscles and occasional twitchings in the face and extremities. Careful observation discloses the following symptoms: "Resistance, rigidity, peculiar tetanoid attitudes of the extremities, and frequently grimacing with the risus sardonicus, irregular twitching and jactations appearing in exacerbations. The reflexes are usually exaggerated, rarely absent." Babinski's reflex was found in one case. No electric changes. The difficulty of coördination soon involves speech and deglutition, and diarrhea often noticed with slight fluctuations in temperature. Condition may last for a few days or a couple of weeks, and if it improves for a time a fatal relapse is bound to occur. On autopsy besides the motor cells, those of the columns of Clarke, of Dieters' nucleus, the central nucleus of the medulla and the nuclei of Goll and Burdach are found involved. Further decay of the myeline sheaths in the pyramidal tracts, the connection of the cortical projection fields with the thalamic nuclei, the fibers of the fillet, the restiform body and the posterior columns of the cord. The changes are different from those in general paresis. The problem is the correlation of the anatomical findings with the important features of the symptom complex and the way in which the condition might be produced. The combination of a rigidity with characteristic attitudes, and perhaps twitchings, is the feature most directly related to the affection chiefly of the cerebral and cerebellar suprasegmental mechanisms. As to the causation, symmetrical parenchymatous affections in the nervous system are usually referred to toxins. Dr. Meyer urges further study of these conditions.

6. *Hydriatic Procedures as an Adjunct in the Treatment of Insanity.*—The case of a girl of eighteen is cited, whose occupation, that of a factory operator, suffering from an attack of acute agitated melancholia of three weeks' duration. Soon after admission she was given a warm pack, which had a marked sedative effect and induced sleep. The warm packs were continued during the first week twice a day for three hours, at the end of which time the patient slept better, bowels relieved and toxic manifestations, which were prominent at the start. This treatment was then discontinued, but in about two weeks mental symptoms returned, accompanied by numerous small furuncles, when a hot air bath of 180° F. for ten minutes was given daily, followed by a needle bath of 60° at 15 pounds pressure for one minute. At the end of the first week of this treatment the mental symptoms subsided, appetite improved, sleep became quiet and restful. At the end of the third week of this treatment she was given tonics, while the Scotch douche at a temperature of 100° F.-59° F. with 25 pounds pressure was substituted for the needle bath. This was continued for three weeks and then discontinued. Patient discharged recovered in four months after admission, having gained 28 pounds in weight. A second case was that of a young married woman of twenty, who became insane after her first confinement. Disease had existed three months prior to admission, when she was dull, depressed and refused to speak, being too confused to answer questions. Refused food, sleep excessive, physical condition fair. Her treatment consisted of a hot air bath of 180 F. for ten minutes, followed by jet douche at a temperature

of 50° with 25 pounds pressure for one minute, and followed by light massage. No apparent mental improvement during the first week, but physical condition bettered. During second week became brighter and more active. At the end of the third week Scotch douche at a temperature of 110°-59° for two minutes with a pressure of 59 pounds, was substituted for the jet douche. She continued to improve mentally and physically. The Scotch douche was continued daily for three weeks, when all treatment was discontinued. Discharged recovered in four months, having gained 30 pounds in weight. A young Russian woman of thirty admitted with acute mania with delirium one week after the onset of the disease, which was ascribed to lactation. Physical condition good. After washing out the stomach she was given a Sitz bath at 100° gradually increased to 112°, with local massage of the abdomen and pelvis while in the bath; was kept in the bath twenty minutes, when she became much quieter, though still delirious. Becoming very noisy and violent six hours later she was given hot full bath at a temperature of 100° gradually increased to 112°. At the end of one hour she showed signs of exhaustion; stimulated, put in bed and heavily covered to continue perspiration; this continued two hours, when she became quiet and slept several hours. On the second day again noisy and maniacal, but delirium not so marked. Hot full bath repeated at same temperature, and continued on three days, when she was much quieter, the delirium had subsided, yet maniacal symptoms continued for two weeks, and received full hot bath daily at bedtime, which procured her good nights. Her symptoms had all disappeared three weeks after admission and was discharged recovered after two months at the hospital. Packs, warm and hot full baths have hypnotic and sedative effect, and are also excellent eliminatives. Sprays and douches have decided tonic effect on glandular action and general cutaneous circulation. No conditions contraindicate hydrotherapy, except advanced pregnancy, pleurisy or when patient is practically moribund. Serious exhaustion is a danger attending warm packs and hot full baths, but this is easily averted by careful observation. Patients who are at first opposed to these procedures soon come to enjoy them. Dr. Dent urges that more attention be given the use of water in the treatment of the insane.

7. *Medico-Legal Phases of the Vermont Observation Law for Criminal Insane.*—"An act authorizing persons indicted for offenses or committed to jail on a charge therefore, whose plea is insanity, be ordered into the custody of the Vermont State Hospital for Insane, to be there detained until the further order of the judge, so that the truth or falsity of such plea may be ascertained. (Vt. Statutes, Acts of the 15th Biennial Session.)" Statistics are given from various sources showing that quite a large percentage of those convicted of crime were insane at the time of its perpetration; the difference between the legal and medical acceptation of insanity is set forth, as well as several cases of individuals cited, who were charged with crime and under observation were found to be insane and others malingerers. This method of procedure does away with the discrepancies in expert evidence. The writer sums up the advantages of such a law in the following words: "This statute stands as a thoroughly conservative and preeminently rational mode of procedure, which from a distinct financial point of view saves the State the usual expense in expert witness fees and lessens the liability of a retrial. It gives the prisoner a careful and impartial medical and physical examination and endeavors to establish a result, which like a final qualitative and quantitative analysis, has been arrived at, through many observations and tests, that owing to the diligence and accuracy with which they have been made, must be infinitely more trustworthy, fair and impartial than would be possible with any set of opinions which have been given from hypothetical questions or the usual short interview. Irresponsible persons are therefore less liable to be convicted. This law is an effectual

damper upon the too frequent plea of insanity, since the State shows itself determined to elicit the *bona fide* character of the plea."

8. *The Possible Influence of Rational Conversation on the Insane.*—A form of psychical treatment that has been found of benefit in some convalescents with periods of depression and excitement, or when the disease insight has just manifested itself and then acts as a support to the patient's own ideas, further, has a diverting influence on the patient's own morbid ideation, but otherwise nothing new is offered that is not the daily experience and mode of procedure of every trained alienist.

McCorn (Amityville).

### ARCHIVES DE NEUROLOGIE.

(Vol. 14, 1902, No. 83, November.)

1. The Pathogenesis of Fundamental Delusional Ideas, Imperative Conceptions and Obsessions, their Affinities to Insane Delusions. A. Paris.
2. Insanity, Communicated and Simultaneous. Guiard and de Clerambault.
3. Moral Idiocy and Particularly of Lying as a Symptom of that Mental Type. Bourneville and Boyer.

1. *The Pathogenesis of Fundamental Delusional Ideas.*—The author states his conclusions founded upon the case of a married woman, thirty-seven years of age. Four pregnancies, last child eight years old. Character emotional, very impressionable; intelligence limited. No important information as to family antecedents, but heredity attested by symptomatology. Manner of living poor; condition one of poverty. No alcoholism. She became a victim of extreme fear of assassination, refused food, manifested more anxiety for her children than for herself and ended by taking them to a neighboring river to drown them with herself. She thought she was accused of crimes and protested her innocence; finally she came to think that perhaps it was in her sleep that she committed the acts of which she was convinced that people accused her, though she did not hear them utter the accusations. She then did her best not to sleep and begged that she might be left without sleep. Later in her history she refused food, stating as a reason that she had no stomach, lungs, *etc.* This delusion, the author thinks, she derived from a fellow patient in the dormitory. On her separation from this patient she freely accepted her food. The author claims that this case "gives evidence, in a way particularly remarkable, on the side of the preponderating rôle of heredity, of original nervous constitution, of insane (Maudsley) temperament in a melancholic, in the genesis of the idea of culpability, the influence of obsession in insane delusion." The case shows, he thinks, "as clearly as possible that the idea of culpability has its inception, mainly, as a consequence of the tendency in some sort innate in the melancholic to self-accusation and shows how the obsession intervenes to produce the formal affirmation, the fixation of the idea of culpability." "It is very evident," he says, "that the ideas of culpability are not simply the consequence of ideas of persecution." "Why, if the rôle of the original defect was not the principal one, if the idea of culpability did not exist previously in, so to speak, a latent state in her, would she not be drawn, like the primary paranoiac, to protest solely to the contrary against the persecutions of which she is the object, and try to demonstrate them unjust, instead of striving, while protesting against them, to establish that she merits them." He states that the ideas of culpability do not arise from hallucinations of hearing, because she did not hear the accusations made, she judged from the gestures and attitudes of the persons about her. "Deductive reasoning has but an insignificant part in the idea of culpability." "It

is evidently the original defect which gives the orientation of the delusion, the obsession, the consequence itself of that defect." The influence of the surroundings of the patient merits a certain attention, since it might have mischievous consequences, as the patient's refusal to take nourishment evidently shows. The author thinks that it is "by attempting to distinguish the delusional ideas, which proceed essentially from the original defect, that we shall come to establish finally a rational classification of insanities properly, of mental defects not attaching themselves to any physical affection, and to determine finally the evolution of each." "After what we have seen in the patient, we cannot conclude that because ideas of persecution appear at first more marked and conspicuous, they are the ones which cause the idea of culpability, but, as I interpret them, they contribute to facilitate their genesis and evolution, the idea of culpability pre-existing in a latent state and thus constituting one of the fundamental elements of the melancholia character prior to the mental alienation. So that, also in the melancholic called the delusion of persecution, the idea of culpability will be moreover, in spite of appearances, a fundamental idea." "The character clearly obsessive of the phenomena which preside at the evolution of the idea of culpability, the intensity, the standing in relief of idea of persecution and the facility with which our patient adopts the delusional ideas of one of her companions, bring me, in conflict with most classic works, to consider the delusion of persecution, or melancholia, as a variety of degeneracy."

2. *Insanity Communicated and Simultaneous.*—There are two cases commented upon. In the first the facts are as follows: the patients are three sisters residing in Paris. They were under the delusion of persecution, said that the crowd in the street cried out: "See them"; called them beavers, monks, the Salvation Army, the Three Sleepers, *etc.* To avoid persecution they first took up a nomadic life in hotels, remaining in each sometimes two weeks, sometimes one, and sometimes only for a single night. They had an income of five hundred francs a month sent to them. For two months they continued the hotel life, and then, to avoid sleeping on benches, they took up their lodging in public hacks. At midnight they would engage a cab by the hour and drive around in it until eight o'clock in the morning. The three occupied the one seat, one sitting in each corner and the younger sitting between the two. They slept well, one, however, by turns keeping watch, and escaped persecution. They made their toilet in the public chalets, ate dry bread, drank at the fountains, passed as much time as possible in churches and museums, and sometimes even walked twelve to fifteen hours a day, and on New Year day, when it rained, they remained standing under portes cocheres. The expense per night was, they said, from sixteen to fifty francs. They spent in some months twelve thousand francs. After an altercation with a cabman, who caused some excitement in the street, they were taken to the Special Infirmary. The sister Annette seemed the controlling spirit. She died at the Infirmary from bacillary infection (not tuberculous). Upon her death the other sisters improved and appeared to be losing their delusions. The authors claim that the delusion had been attacked at its source by change in the environment of the patients and by partial isolation, which had been instituted. *Case II.*—Insanity communicated from mother to son. Psychosis of long standing in the mother. Transmission to son of maternal delusion. Genesis in son of second psychosis independent of the first. The mother of the age of sixty-two years was brought to the Infirmary on March 25, 1902. Her volubility, multitude of ideas, of reasons, of proofs indicated a delirium of long standing. She was married at twenty to a mechanic; became a widow; at twenty-six married a park keeper. She

adored him and before marriage wrote him several letters each day. For a time her happiness was perfect, then came the war, the siege and her husband, sent out one day to bear dispatches, disappeared, shot by the enemy or drowned in the Seine: it was never known. She remained a widow and was compensated by a *bureau de tabac* and a small pension, and lived in Paris with her three sons. Two died while still young. Her grief, the mystery which surrounded the death of her husband, an event which had greatly shocked her, revived her former love of writing on topics social and philosophic. She wrote the "Drama of my Life" and the "Planetes Rocheuse" and became possessed of the delusion that people were trying to seize her writings and enrich themselves by their sale, and Flammarion had plagiarized them. She lived very retired, her pension and shop bringing her but 1500 francs a year, and she expended considerable sums in posters proclaiming her wrongs. Her son was brought to the Special Infirmary at the same time. He was of the age of thirty-seven years, artist painter and could sell none of his works. He partook of the delusions of his mother, had various original ones about Dreyfus and the Transvaal war, that people spoke evil of him to prevent him gaining his living. He attempted suicide, necessitating his removal to the Infirmary. In résumé, the delusion communicated by the mother to the son had this peculiarity, that that of the son was the reproduction complete and absolutely passive of that of the mother. Then the special delusion of the son diverging, evolving on its own account upon a soil all prepared.

In the first case the delusion came simultaneously to the three sisters under the influence of the same causes, but the part of each of them in the association is not equal. The younger is manifestly the most active, the most suspicious. Her mind is always strained and distrustful and it is clear that she leads in the delusion. the two others play a more subordinate rôle. In the second case the delusion is communicated by a mother to her son, a being weak and subjugated; he is quite truly insane and one should not declare that he will be restored to reason, the gravity of the prognosis being drawn from the soil upon which the psychosis has been developed. One sees that ideas of persecution play the principal rôle in these two cases; it is in effect the rule. The idea of persecution develops itself easily in an environment of depressing conditions of all sorts, and above all from poverty, and *folie-a-deux* is especially the lot of the unhappy; one understands, without its being necessary to insist upon it, that two unfortunates, ruined by privations in identical conditions exceptionally favorable to build up in common a delusion of persecution, or to consider as true a delusion of that kind engendered solely by one of them. We may then conclude that the division into folie communicated and folie simultaneous is legitimate and corresponds to the reality of fact. Useful for grouping in nosography, it presents still this very appreciable advantage in psychiatry and precise terminology. If it does not always accommodate itself to the diversity of the clinic, for here as elsewhere, there are some intermediary cases. We should not forget besides, according to the first remark of Laseque and Felret "that it deals with one of the forms of alienation, intermediary between reason and insanity, and which exempt from marked physical troubles, only lend a psychological analysis. The cases naturally derive from the malady an aspect quite peculiar and resemble rather studies of manners than medical observations."

3. *Moral Idiocy and in Particular of Lying.*—This is a new observation relative to lying by children attainted by moral idiocy, which completes in divers respects those published by Bourneville and Boyer in the Archives, 1902, No. 76. The authors set forth in considerable detail an account of the accusation by a girl of fourteen against her father, charging him with violation of her person. She was proven thief and liar. An examination showed the hymen intact. Her hereditary taint is sufficiently

manifest. Father has violent headaches with vertigo; paternal grandfather alcoholic, epileptic, demented, died in hospital; mother affected with cardiac troubles and probably exophthalmic goiter. We have a case of moral idiocy characterized by the perversion of the instinct of veracity and of that of property (lies and thefts). If she had not made up a lie out of whole cloth, she had put an interpretation consciously false upon acts little reprehensible in themselves. The genesis of this monstrous lie was in part explained by the imprudence of the father, who lived with a mistress, who had become alcoholic and lived from prostitution. The authors show the danger of accepting too literally the testimony of a child. Under medico-pedagogic treatment she notably improved. She manifested a desire to become a nurse; attempts to that end having failed, she was placed as a domestic in a family of the middle class, where so far she had been perfectly well behaved.

RICHARDS (Amityville).

(Vol. 14, 1902, No. 84. December.)

1. Obsessions in Mental Pathology. ATHANASSIO.
2. Insane Automutilator. POIRSON.

1. *Obsessions in Mental Pathology.*—A foot-note states this article was a paper presented to the Academy of Medicine and that it received *"mention honorable au Prix Civrieux."* The author contributes a treatise on the subject of obsessions, of which 16 pages are given in this number and of which there will be a continuation in one or more subsequent numbers. It is not very capable of abstraction, except in the sense of stating the points discussed. He first furnishes a definition of obsession from the pathological point of view. According to Magnan "obsession is a mode of cerebral action in which a word, a thought, an image imposes itself upon the mind, in spite of the will, but without distress in the normal state; with a painful anxiety, on the contrary, which renders it irresistible in a pathological state." Our author gives the different names under which it is described, as emotional delirium, mania without delusion, monomania, fixed ideas, imperative conceptions, uncoercible ideas, abortive delusion, rudimentary paranoia and obsedant ideas. Classification by Regis: (1) The state of diffused anxiety or panophobia; (2) The state of systemized anxiety or monophobia; (3) The state of anxious idea or monoïdeic. Freud admits rudimentary attacks of anxiety, which may produce: (1) The respiratory type; (2) The cardiac type; (3) The sweating type; (4) The trembling type; (5) The type of voracious eating; (6), The diarrhea type and of frequent urination; (7) The vasomotor attack; (8) The paresthetic attack; (9) Pavor nocturnus; (10) Vertigo. The author discusses monophobias or phobias properly so-called and obsession properly so-called. "The nature of the ideas of obsession are eminently variable. Among 250 cases of well-defined obsession, Regis found especially: the obsession of being insane, of itch, syphilis, cancer, apoplexy, sudden death, pathological softening, general paresis, a foreign body in the ear, microbes, contact of physicians, heart disease, blenorrhagia, madness, vertigo, aphasia, verbal amnesia, ataxia, fainting, pregnancy, *etc.*, *etc.*, the religious and scrupulous obsession, with its infinite varieties (anxious idea not to touch a person or thing having been in contact with the consecrated wafer, in particular priests and those who take communion, for fear of moral pollution, and the obligation to wash constantly the hands as in the obsession of physical contamination)." "The most common of all these obsessions are those of a religious form. The patients wish to pray, to make a confession, then comes to them a blasphemy, an impiety, a sacrilege, a gross insult in thought or in speech. At other times the subjects are constrained to contradict themselves, to say just the contrary of that which they think or of that which they would wish." "Regis and Pitres give as a

general character of obsessions: to produce themselves in the morning on awakening, at the passing of the dream life, accompanied most often by the momentary forgetfulness of their moral torture, at the reappearance of real life, this being with many, as with a number of neurasthenics, the worst moment of the day. Others are taken every evening at nightfall with distressing paroxysms." "Sleep is more or less good. Sometimes the obsession has no repercussion upon itself, at other times it occurs in the dream." "In all cases, in the intervals of the crises, when they are not too intense, the subjects are able to pursue their calling. Habitually they conceal their state of mind and concentrate themselves on themselves, even avoiding speaking of it to their nearest friends. It is only when they are at the end of the strife or too much tormented that they confide in the physician, obtaining in that confession, as do neurasthenics, a temporary relief." Regis classes obsessions as constitutional and accidental. The first with hereditary taint are precocious, the intellectual element predominates, they have a mode of conduct remittent or continuous, are chronic; the obsedent idea is multiple or may modify itself. With heredity less strong, mainly in the point of view of insanity, the onset more tardy, the preponderance of the occasional cause, the production of a phase of phobia, the persistance to a marked degree of emotional phenomena, the conduct always unstable, in fine, its curability are the characteristics which belong chiefly to accidental obsession. (To be continued.)

2. *Insane Auto-Mutilator.*—Poirson discusses the mental state of the patient and also the pathological incidents which supervened in the course of the malady, whilst the subject was at the asylum of Mareville. He first considers the pathological incidents. The patient was thirty-seven years old on admission to the asylum. He was a glazier and day laborer, married, and the father of three children. He was reported as maniacal, jealous, violent. He had delusions of persecution and saw and heard a virgin in white. Six months after admission the discovery was made of a swelling of the cellular tissue over the left pectoralis major. The swelling has a small opening in the center from which flows an extremely fetid pus and of a blackish color. An incision and cleaning produce besides a large amount of pus, a match, the lead of a pencil 5-6 cm. in length and a fragment of the wood of a pencil. In spite of careful attention, the swelling extended in every way. It was diffused over all of the left thorax with large blebs. It caused marked general debility. The treatment consisted in putting to bed, multiple incisions, drainage, irrigation, antiseptic dressings. Two weeks later the purulent discharge, very abundant till then, diminished a little and in the course of the next month the swelling healed and cicatrized in the superior part. Below an oozing of pus and serum continued. The general condition became satisfactory at that time. Later another tumefaction appeared at the level of the upper third of the sternum. An incision was made; pus and serum were discharged and at each inspiration air entered by the wound and at expiration a mixture of pus and serum was discharged of frothy form at the cutaneous orifice. A year after the swelling above described had healed the patient presented a temperature of 39° C. The third and fourth day auscultation revealed tubular breathing of muffled timbre on the left side; percussion of the lungs negative, as well as of the precordial region. The pulse normal at first, but became weaker, then thready, almost imperceptible the last two days. The patient's unfavorable symptoms appeared on April 6th and he died on the 16th. The symptoms observed did not permit the diagnosis of pneumonia, yet the autopsy disclosed that it was a pneumonia, which should have been the diagnosis. The signs given by the percussion, *etc.*, were negative, because of the localization of the disease in the center of the lung. As to temperature pericarditis explains the abnormal character of the temperature during the pneumonia. There was moreover found

in the left lobe of the liver on its upper surface a pin without head, implanted in the hepatic tissue. The liver of normal size, but had the aspect and yellow color of infectious and cachetic degeneration. As to the patient's mental state, indicated by his self-mutilation, "under what inspiration did he act and what was his object?" No satisfactory replies were obtained from him. "Auto-mutilators most often obey their delusional ideas. Some wish to punish themselves for crimes they believe they have committed, others think to escape from persecutions; sometimes they are simply the result of despair, often the motives are incomprehensible." "In our patient by reason of his delusion, essentially polymorphic and in absence of all indications, the exciting cause is alone known to us, *i.e.*, blunting of sensation." "In that state did T. perhaps obey a voice? Did he wish to deliver himself from some evil spirits within him? Did he wish to punish himself for some faults, of which he believed himself culpable? As to the efficient cause of his mutilation we make any of these suppositions."

RICHARDS (Amityville, L. I.)

### NOUVELLE ICONOGRAPHIE DE LA SALPETRIERE.

(15th year, 1902, No. 6. November-December.)

1. On the Affections of the Cauda Equina and of the Inferior Segment of the Spinal Cord. F. RAYMOND.
2. Syphilitic Lesions of the Nerve Centers. Hemiasynergia, Lateropulsion and Bulbar Myosis with Hemianesthesia and Crossed Hemiplegia. BABINSKI AND NAGEOTTE.
3. The Historical Lesions of the Cortex in the Atrophies of the Cerebellum. LANNOIS AND PAVIOT.
4. The Protoplasmic Prolongations of the Nerve Cells of the Horns of the Spinal Cord in the New-Born. SOUKHANOFF AND CZARNIECK.
5. Gigantism and Infantilism. LAUNOIS AND PIERRE ROY.
6. The "Paotred ar Zabat" Breton Legends. DUCREST DE VILLENEUVE.
7. Giants in Art. HENRY MEIGE.

(1) *Affections of Cauda Equina.*—This is a further study of the inferior segment of the spinal cord, which has of late been the subject of several communications by Raymond. The cauda equina includes the total number of nerve roots which arise from the lumbar enlargement and from the conus terminalis, and the fibers which go to form the sacral and sacrococcygeal plexus. At the present time the term "affections of the cauda equina" is applied to pathological cases which are distinguished by the partial or total integrity of the sensory motor functions of the inferior extremities which are innervated by the lumbar plexus. To assume that a morbid process affects the total number of roots of the cauda equina, it is necessary to include, among the other symptoms which may be present, a total sensory and motor paralysis of the inferior extremities. The location of the lesion, whether nuclear or radicular, is of great importance. Three cases are quoted to illustrate the diagnostic principles which are to be drawn upon in localizing the lesion.

*Case 1.*—Woman, aged thirty years. Two years before, without any known cause, other than fatigue due to the patient's profession, she became the subject of violent pains extending to the buttocks and thighs. They became more intense on the left than on the right and followed in general the distribution of the sciatic nerve. Difficulty in locomotion soon followed, and bladder incontinence with retention soon developed. Progressive atrophy of the muscles, more marked on the left than on the right, disappearance of tendon reflexes, and an anesthetic area which at first occupied the internal aspect of the buttock on either side, soon made their appearance. The plantar surface of the left foot and the extension of the area of anesthesia to the posterior surface of the leg completed the picture. The seat of the lesion was believed to be in the nerve roots composing the cauda equina and not in the cord segment which gave rise to them, for these reasons: The excentric direction in which the violent pains were propagated, the unequal intensity of the pain on one side as compared to the other, and the bladder disturbances. Raymond believes this to be a neuritis, implicating the nerve roots and produced by the fatigue which the profession of the patient caused. The patient was a mechanic by profession. The prognosis of the neuritic variety is more favorable than that of the nuclear.

*Case 2.*—A man thirty-eight years old, alcoholic. Paresthesia of the left lower extremity, followed by a motor paresis, developed after exposure to cold; incontinence. Four months afterward the condition was as follows: motor paresis very pronounced of the muscles of the posterior aspect of the thigh; the leg muscles and the left foot. Atrophy and R. D. in the region supplied by the external popliteal branch of the sciatic. Abolition of the tendo Achillis reflex. Intermittent pains along the sciatic region; anesthesia of the left half of the scrotum, penis, perineum, and of the adjacent buttock territory and of the thigh, in the form of a band, the size of which diminishes gradually from top to bottom. The anesthesia extends to the external portion of the dorsal aspect of the foot; abolition of anal reflex, suppression of erection. The diagnosis of a neuritis of the nerve roots of the cauda equina, with a relatively favorable prognosis was made.

*Case 3.*—Man, age twenty-eight, motor paralysis of the left lower extremity, atrophy of the muscles of the posterior aspect of the thigh and of the calf, with a lowering of the local temperature in these regions. The knee jerk and the Achilles reflex abolished. On account of the acute onset of these symptoms, with temperature, and the sudden paralysis of both extremities, followed by a permanent paresis of the left one, the rapid appearance of atrophy, the coldness of the extremity, the disappearance of both tendon reflexes, led the author to make the diagnosis of hematomyelia, or rather of a hemorrhagic poliomyelitis limited to the anterior column of the spinal cord segment which gives rise to the nerve roots which form the sacrococcygeal plexus.

2. *Syphilitic Lesions of Nerve Centers.*—Babinski and Nageotte presented to the Neurological Society at Paris, April, 1902, a description from a clinical point of view of a disease closely allied to a unilateral bulbar lesion. Three cases formed the basis of this communication. The essential features were hemiplegia, hemianesthesia on the side opposite to the lesion, hemiasynergia, lateropropulsion, and myosis on the same side as the lesion. One of these cases came to autopsy, and this paper is given up to a further study of this disease, especially from the anatomical point of view. A man fifty years old, syphilitically infected at the age of thirty, was attacked suddenly while in perfect health with hemiasynergia limited to the inferior left extremity, lateropropulsion towards the left, slight tremor of the upper extremities, a moderate degree of hemiplegia and anesthesia on the right, difficulty of deglutition and slight narrowing of the left pupil. Twelve days after the attack the patient died and the autopsy showed the presence of arterial syphilitic lesions and diffuse meningitis with multiple foci of softening located in the left half of the medulla. From these clinical and anatomical data, the authors have felt justified in drawing certain deductions relative to the mode of development of syphilitic affections in general and to the relation which exists between the observed symptoms and the localization of the morbid processes as determined by microscopic examination. Some of these deductions are the following: Generalized meningeal lesions appear to be the rule in a nervous syphilis during the process of evolution whatever its form may be, whether tabes, general paralysis, or multiple lesions of the brain and cord. These

lesions constitute in some way the foundations upon which are based the further developing lesions characteristic of every form of syphilis of the nervous system. The lymphocytosis which is found in the cerebrospinal fluid preceding the appearance of the meningeal symptoms, appears very early. It accompanies the first symptom of nervous syphilis and may be its only manifestation. In consequence, a meningitis appears to be the first stage of any lesion of the nervous system which is the seat of a syphilitic infection. Syphilitic meningitis bears a close resemblance to tubercular meningitis from the histological point of view. The differentiation is to be made by the presence of a less discrete cellular infiltration and the absence of true caseation in the former as compared with the latter. The authors, however, confess that the distinction cannot always be made. In the case here described the diffuse arterial lesions have determined the areas of softening in the medulla, upon which the symptoms depend. The lesions caused the following interruptions in the nerve tracts: (1) An interruption of the olive-ciliary pathway of both sides. The cells of the inferior olivary bodies of the medulla are not in connection by means of their axis cylinders with the cells of the cerebellar olive and of the nucleus emboliformis; (2) An interruption of the descending path which connects the nucleus of Deiters on the left (perhaps the left hemisphere of the cerebellum), with the left side of the medulla; (3) An interruption of a portion of the fibers of the fillet; (4) An interruption of the lateral ascending paths of the cord (Gowers' column), with a conservation of the direct cerebellar tract, probably; (5) A lesion of the longitudinal posterior tract, left, with a descending degeneration; (6) Destruction of mixed nerves on the left side over a certain extent; (7) A slight lesion of the left pyramid.

3. *Histology in Atrophy of Cerebellum.*—In 1901 the authors, Lannois and Paviot, made a preliminary communication to the Society of Medical Sciences in Lyons in which they showed that, in certain cases of atrophy of the cerebellum, the cells of Purkinje and the cells of the molecular layer were found absent and a new layer of cells appeared in their place, which had not yet been described by histologists. They decided that these cells were present in the normal cerebellum lying between the molecular and granular layers, above the layer of the cells of Purkinje, and that they could be identified with the large cells described by Cajal, Golgi, Bechterew, and called by Dejerine large cells of type II of Golgi. They have been able to study three additional cases of cerebellar disease which throw light on this subject. They are here reported, together with the results of the anatomical studies. *Case 1.*—Cerebellar syndrome with muscular asynergia of the trunk and extremities. Cerebellar atrophy apparently limited to the base, a subcerebellar pseudo-cyst. Chronic diffuse meningitis. *Case 2.*—Epilepsy. Attacks very frequent, with a sensory aura. No loss of consciousness. Atrophy of the left lobe of the cerebellum. *Case 3.*—Spasmodic infantile atrophy on the right. Epilepsy. Cerebral sclerosis of the left hemisphere, with crossed atrophy of the cerebellum. In these three observations of primary or secondary atrophy of the cerebellum a lesion has been found which, to all appearances, is absolutely characteristic. The lesion appears in the convolutions of the cerebellar cortex. The Purkinje cells disappear absolutely without leaving the least vestige of degenerative cell bodies in the process of destruction. The cells are either normal or are not seen at all. At the same time the granular layer lessens, then disappears, and shows by the Nissl stain no trace of cell structure. Together with the double disappearance of the normal cell layers of the cerebellum, there appears outside of the granular layer, and sharply separated from it, a layer of oval and pale cells which have a nucleus of chromatin and from one to two nucleoli, which are very deeply stained. This new layer does not give the impression of having been newly formed. It seems to

have existed before under the Purkinje cells and those of the granular layer, and it only comes into evidence with the disappearance of the two former. That this layer has not been before recognized is due chiefly to the method of staining used, as in the Weigert-Pal preparations it appears very faintly. Thomas and Dejerine have probably seen them, judging from the description which is found in their well-known work, Le Cervelet, 1897, but they failed to give to them any great importance. The following conclusions end the article: (1) There is no histological difference between the cortex of the cerebellum, which is primarily atrophied and one which is secondarily atrophied; (2) In the process of atrophy there becomes isolated invariably a layer which corresponds probably to the cells which lie between the granular and the molecular layer (cells of Type II, Golgi). The molecular layer atrophies at the same time that the cells of Purkinje and the granular cells disappear. The disappearance of the cells of Purkinje is total and absolute and precedes all other changes in the atrophic process. The frequently occurring secondary nature of the atrophies shows that one should not consider cerebellar syndrome as being always the type of the primary lesion of the cerebellum.

4. *Nerve Cells in Horn or Spinal Cord.*—In a previous communication *Le Nevraxe,* (see Jan., 1903, No., JOURNAL NERVOUS AND MENTAL DISEASE), the authors of this article have demonstrated the possibility of using the chrome-silver method in the study of the adult spinal cord. They made sections in a longitudinal direction parallel to the central canal. In this way they were able to study with great accuracy the protoplasmic prolongations of the nerve cells. The present paper is concerned with the appearance of these prolongations in the cells of the anterior and posterior horns in the cords of newborn infants. Their method is as follows: Small pieces of cord were cut in half in a dorso-ventrical direction and placed in a chrome-osmic mixture for four days. The specimens were then placed for two or three days in a 2 per cent. solution of silver nitrate to which was then added osmic acid. Some of their results were as follows: There exists a very marked difference between the aspect of the protoplasmic prolongations of the cells of the anterior and those of the posterior horns. In the former the dendrites are more regular and come off more at right angles. They are also very poor in collaterals. In the posterior horns the dendrites are short, more ramified, their contour is less regular, and they are richer in collaterals of a varying form.

5. *Gigantism and Infantilism.*—Launois and P. Roy say that gigantism and infantilism are two anomalies of evolution which, at first sight, appear to be so different that any attempt to bring them into the same category would seem impossible. A relation between them was suggested by the study of some cases scattered in the literature and by the observation of a case upon which this article is based. The case here reported is that of a man of large stature, measuring in height two meters and four mm. Various stigmata of infantilism can be demonstrated. The man continues to grow, although he has reached the age of thirty years. A careful description is given, together with measurements, photographs, and radiographs. Other cases are cited, and from the data thus obtained these conclusions are reached: (1) There exists a type of infantile gigantism in which large stature coexists with genital atrophy, or at least with impotency and sterility, facts which have been noted among giants; (2) This type of infantile gigantism, especially in regard to the continuity of growth (persistence of cartilaginous union), to the manner of this growth (increase in length of extremities and especially of the inferior extremities), and in regard to its anomalies (genu valgum), is not always perhaps to be considered as belonging to the type of acromegalic gigantism (large body, hypertrophy of the extremities, deformity of the inferior maxilla, *etc.*), because the retardation in the ossification of the epiphyseal cartilages

and the hypertrophy of the hypophysis are common to both; (3) It remains to be determined the part that the pituitary hypertrophy plays in this production of abnormal growth. In this regard the examination of the hypophysis in eunuchs and in animals which have been castrated early in life, can perhaps furnish us with the desired information.

6 and 7.—Articles of literary interest solely.

SIDNEY I. SCHWAB (St. Louis).

### MISCELLANY.

TREATMENT OF GRAVES' DISEASE. E. Schultes (Münch med. Woch., May 20, 1902.)

The author has made a series of investigations on the action of a serum obtained from sheep or dogs from which the thyroids have been removed, following the initial suggestion of Baller and Enriquez. He reports the case of a woman forty-nine years of age who had had exophthalmic goiter four years. Palpitation, anxiety and delirium were prominent symptoms. The exophthalmos was marked, the thyroid much enlarged, circumference of neck being 17 inches. The initial dosage of this new body, prepared by Merck and termed antithyroidin, was gm. 0.5 (7.5 grs.). This was increased until she was taking gm. 4.5 (70 grs.) three times a day. After two weeks her delirium was markedly improved, the pulse rate has dropped to 100, the circumference of the neck diminished almost an inch. A week later the pulse rate was 88. The thyroid was softer and the patient was able to attend to light work, such as sewing from which she had been deprived by reason of the marked tremor. After seven weeks she was sufficiently better to be termed well.

NEURASTHENIA. Joseph M. Aiken (Medical News, Nov. 29, 1902.)

Neurasthenia is a primary affection of the nervous system, especially common in individuals with hereditary defects. The unstable constitution is a neurasthenic in latency, needing only some shock, moral or physical to develop neurasthenic symptoms. Diminished dynamic energy, and lessened recuperative power in nervous structures is the chief pathological condition. Impaired metabolism with accumulation of waste products, which in turn accumulating in the blood, give rise to auto-intoxication, especially affecting the nervous system. Deficiency, never loss of function and fatigue, are common symptoms, but no loss of sensation. Headache is never absent, or pains in the back or limbs. Diminished sexual, visual or digestive power are common. Mentally, weakness or irritability are common.

Treatment should be hygienic, dietetic and medicinal; with special stress on strange environment, careful hydrotherapy, sleep, exercise and diet strictly regulated by the physician. Hypnotism still farther exhausts weakened nerve power. Water should be given freely, except at meal times, often hot.

W. B. NOYES.

SYMMETRICAL GANGRENE (RAYNAUD'S) *versus* ENDARTERITIS OBLITERANS James Dudley Morgan. (The Journal of the American Medical Association, Nov. 29, 1902.)

The smaller blood vessels seem to be affected first in Raynaud's disease. They have more muscular tissue in their middle coats, and are thus more capable of active changes in caliber, and this muscular tissue is under control of sympathetic nerves, which nerves are controlled by sympathetic nerves, which nerves are controlled through their center in the spinal cord. Friedländer's disease, or obliterating endarteritis, generally shows neuritis. Raynaud's gangrene may occur without neuritis. There is an abnormal excitability of the vasomotor centers in the spinal cord, producing a spasm of the vessels. True Raynaud's disease is rare. It should not include cases with proliferation of cells of the intima. Both diseases occur frequently in middle life. Syphilis is not a special cause of Raynaud's disease. Valvular defects, congenital narrowing of the aorta and arteries are not infrequent causes. There is a similarity between hemoglobinuria and Raynaud's disease in that exposure to cold produces attacks. Heredity is found in 8 per cent. of the cases. A history of exposure to cold, rheumatism, malaria, or tuberculosis is at times found. Less frequently other disturbances of the nervous system, such as hysteria, organic nervous disease, or insanity. A case of Dehio proves that Raynaud's disease can occur as an independent and typical disease in persons otherwise healthy. A healthy woman developed it after a severe fright. The vessels showed a fibrous endarteritis and endophlebitis. Senile gangrene is typically unilateral. Glycosuria cases are easily excluded. Erythromelalgia seldom leads to gangrene, is painful and unilateral. Of two cases reported by the writer, the first showed extremely calcareous arteries; the second showed anterior and posterior tibial arteries completely occluded by a growth of connective tissue, continuous with the tunica intima, though different in character. No real thickening of the tunica intima. Smaller vessels, however, showed obliterating endarteritis. But disease of the arteries had not caused the gangrene, which had begun with a simultaneous and acutely sudden onset. Raynaud's disease never causes death. Why it should cease is a mystery. It may be due to a transient toxic, or reflex agent, having itself only an intense but transitory stimulation of the vasomotor center.

Wm. B. Noyes.

A Clinic on Nervous Diseases. Daniel R. Brower, Rush Medical College (Medical Standard, Dec. 1902).

1. *Epilepsy.*—A matter of considerable interest is the epileptic habit in old epileptics. If the patient has an aura the habit may be treated with reasonable chance of success by the inhalation of amyl nitrite. For if this is inhaled when the aura appears the fit cannot come on. If the aura is in an extremity, a counterirritant may be sufficient. All epileptics are intensely emotional, so any line of treatment must be accompanied by the positive assurance that it will do good. The author uses bromides in small doses, avoiding bromism, and believing that it is better for the patient to have some seizures and a clear head between, than to be in a state of mental incapacity from bromides all the time. Sodium bromide is less disturbing to the digestion and less likely to cause anemia than the other bromides, but in any case an iron preparation, such as Basham's mixture, should be administered. In almost all epileptics there is a feeble, easily compressed pulse with cardiac insufficiency which may be treated best of all by adonis vernalis. The bromide dosage may be quite small if accompanied by the fluid extracts of horse nettle and of solanum carolinense, zfs-ij (cc. 2.-8.). A laxative pill and an intestinal antiseptic, as salol, may also be considered routine treatment.

2. *Tabes Dorsalis.*—In the pre-ataxic stage avoid strychnine and prescribe rest, moderate doses of opiates, and tonics, preferably phosphorus, iron and arsenic. A mild but efficient alterative that does not disturb the stomach is resin guaiac, gr. iij (gm. 0. 2.), and chloride of gold and sodium gr. xx (gm. 1. 3), a half hour before meals; another preparation of great value and recommended by Niemeyer is Blaud's pill, one three times a day, with a laxative to overcome the constipating effect of iron.

W. A. Bastedo (New York).

Obsessions: Fixed Ideas, Etc. Theodore Diller. (The Medical News, Nov. 22, 1902).

Obsession includes a group of mental symptoms more or less allied, occurring in various psychical states, which have been called by various writers fixed ideas, impulsions, abulias, imperative conceptions and phobias. They are held to be expressions of neurasthenia, though not exclusively so, many being more closely associatel with melancholia, hysteria, epilepsy, or physiological degeneration, while some seem to occur in individuals otherwise normal. Obsessions may be of five classes: (1) Of doubt; (2) of fear; (3) of impulse; (4) of miscellaneous ideas; (5) abulias. Chemical histories of 17 cases were given:

Case I, fear of disorder or dirt, was cured by bluntly disregarding them. Case II, suicidal obsession, which finally caused patient to be sent to an asylum. Case III, hypochondriacal obsession, an imagined condition of "fermented stools." Case IV, the fear of definite or general personal danger, in a subject with stigmata of degeneration. Case V, fear of public speaking. Case VI, stuttering when tired or bored with any subject. Case VII, obsession of impending danger, with constant mental depression. Case VIII, believed he constantly smelt of feces. Case IX, believed lice were on her head. Several other cases had obsessions associated with masturbation. The treatment should be directed towards maintaining the physical tone, and impressing the individual mentality. The drug treatment should be as simple as possible. W. B. NOYES.

HEREDITARY SYPHILITIC TABES DORSALIS. Babinski. (Soc. Med. des Hopitaux, Oct. 24, 1902.)

This observer, in discussing the question of hereditary syphilitic tabes dorsalis, said that there are in literature 20 well-authenticated reports of this condition. He thinks that the disease exists more commonly than is generally supposed, probably because it is usually present in a more or less masked form. For this reason he begged to present the histories of two such cases. The father of each of them was himself a tabetic. The first case was a young woman, twenty-two years of age, presenting the teeth of Hutchinson, who up to his eighteenth year had enjoyed good health,excepting that at her birth she presented ulcers around the anus, and that during her seventh year she was seized with convulsive tic. In her seventh year she presented interstitial keratitis, with all the characteristics of hereditary syphilis. During the past two years she has been a victim of crises of pain, and her pupils are not active to light. Further, it is to be noted that the father of this poor patient contracted syphilis while her mother was pregnant with her, and thus herself, became contaminated. The father at present presents the characteristic signs of tabes dorsalis, namely, abolition of the reflexes at the knee and ankle, irritability of the bladder and lightning pains and the Argyll-Robertson pupil. The other patient was a fifteen-year-old girl whose pupils were insusceptible to light, whose reflexes at the knee and ankle were abolished, whose bladder was showing symptoms, who had a choroiditis on the left side of syphilitic character, and, finally, whose cephalorachidian fluid disclosed lymphocytosis. The woman also presented mental disorders pointing toward an early dementia, perhaps due to a meningo-encephalitis of uncertain distribution. Her father was the victim of tabes dorsalis, precisely like the father of the foregoing case. The recognition of facts like these are of really practical value, because it is already admitted that treatment with mercury, energetic and persevering, may exercise upon these hereditary syphilitics a curative effect no less active than in acquired tabes, that is to say, that it may delay in a fixed measure the progress of the disease. JELLIFFE.

RELATION OF CONSCIOUSNESS TO THE NERVOUS SYSTEM. Alex. E. Gibson. (Medical Record, Nov. 22, 1902).

The nervous system is two-fold—cerebrospinal and sympathetic; the former related to the instinctive or psychic processes, the latter to the vegetative or vital, while both are mainly below the sphere of self-consciousness. The sympathetic nervous system carries on its functions without the necessity of cerebrospinal interference and is responsible for the rhythmic movements characteristic to the functions or tissues it supplies. Embryology teaches that the sympathetic system is an outgrowth of the cerebrospinal. Every functional activity of the body is a type of the mechanical principle of the lever notably such activities of unstriped muscle as the pyloric valve, the heart, or sweat glands. It follows that two factors must be present: the protoplasmic storage batteries of vital force, and an elaborate system of conductors. The nervous system holds bodily tissues in a state of constant tension. The evolution of self-consciousness as manifested in a human being is *sui generis* and not demonstrable as a product of functional or structural operation. Self-consciousness seems to be superimposed on the whole biological output of the evolutionary processes. No process of evolution can be traced as in the evolution of the sympathetic as an outgrowth from the cerebrospinal system. The cerebrospinal system, while controlling the sympathetic, is itself operating under the cover of instinctive consciousness and below the reach of individual self-consciousness. The individual has in his charge a mechanism over which, to a large extent, he has no control. The basis of vegetative life as represented in the smooth, non-striated, spindle-shaped, "involuntary" muscle cell, which receives the innervation of the sympathetic nerve, has a common significance to man and animal. This is true also of the striated muscle cell, innervated by cerebrospinal nerves. In the cranial structures in the cerebral cortex the anatomical distinction between animal and plant arises, and as this is chiefly self-consciousness, it is logical to associate self-consciousness with an advanced evolution of the unique, non-demonstrable functions of the cortex. These unexplored regions of the cortex may form the structural basis for the self-conscious existence.

W. B. Noyes.

Perforating Ulcer of Foot. E. Tomasczewsk (Münch. med. Woch., May 13, 1902).

It has been the observation of this writer that ulcer of the foot occurs when sensibility is intact and no trauma exerted on the feet. Ulcers of the feet are well known to resemble each other irrespective of cause, in their form, development and course, and are generally found to occur where the most pressure is exerted. They are preceded by a hyperkeratosis, exerting still more pressure, which leads to circulus vitiosus. The observance of these general conditions has led to the usually accepted hypothesis that a decrease of sensibility in the part affected had caused the patient to pass over the fact that pressure was being exerted by an ill-fitting shoe or other cause. The author, however, advances the theory that an isolated sclerosis of the plantar arteries is largely responsible, as there is often little bleeding during operation, and an absence of pulsation. He has also noted that ulcers seldom occur on the heel, which is supplied by blood vessels of a larger caliber than the rest of the foot. In such diseases as diabetes, syphilis and various nervous disturbances arteriosclerosis is a common lesion, frequently most developed in the parts farthest from the heart. It is not clearly evident whether any trophoneurotic disturbances are at work or not, as the existence of trophic nerve end centers is still unsettled. Degeneration of the nerves which supply the part affected has been shown, however, and these also occur in cachectic individuals without ulcers, or in cases where ulcers are artificially produced. If an examination of the urine does not reveal sugar and the anterior and posterior tibial pulses are negative, then it is well to take a complete nervous status, as the ulcers may very likely prove a subjective manifestation of cerebral, spinal or peripheral disease.

Jelliffe.

# Book Reviews

A PRACTICAL MANUAL OF INSANITY FOR THE MEDICAL STUDENT AND GENERAL PRACTITIONER. By DANIEL R. BROWER, A.M., M.D., LL.D. Professor of Nervous and Mental Diseases in Rush Medical College, *etc.*, and HENRY M. BANNISTER, A.M., M.D. W. B. Saunders and Company, New York, Philadelphia and London.

IN the preparation of this manual the authors have considered before other things two requirements. They have appreciated the needs of the untaught student for sound, readable and yet authentic fundamentals which, augmented by subsequent observation and reflection, will fit him for the recognition of disordered mental states, and secondly, they would place in the hands of the general practitioner some work of ready reference that would be of service in his daily round.

The purposes here outlined have been most admirably carried out.

The opening chapter on "definition" is a most excellent one, and presents the subject in a thoroughly modern and common sense fashion. Recognizing the necessities for some sort of definition for the sake of legal terminology alone, if may be, the authors, after giving the many reasons why insanity is incapable of definition by any terse series of generalized statements, offer the following: "Insanity is a more or less permanent disease or derangement of the brain, producing disordered action of the mind in such a manner as to put the subject in a condition varying from his normal self and out of relation with his environment, and" in general, "in such a way as to render him dangerous or inconvenient to himself or to others."

For legal requirements this definition may be considered satisfactory, but the authors recognize its vagueness and incompleteness from the medical point of view. They do not attempt a medical definition which we believe should be done with the distinct statement that insanity legally and medically requires different view-points and should not necessarily be brought into correlation; furthermore the necessity of considering insanity not as a single entity, but a vast horde of conditions at times entirely unrelated save in their essential feature of an affection of a single complex bodily organ, needs to be brought out more clearly than we here find it.

If there is any general criticism that we would offer it would lie along this line, that the authors have generalized too widely in their chapters on etiology and pathology, believing that it is better to clearly differentiate certain groups of insanities and show their essential etiological and pathological factors. At the present time we do not speak of the pathology of lung diseases, but sharply differentiate, different groups of these affections with allied etiological and pathological features. We believe that the careful studies of the insanities that have been made during the past decade allow similar dividing lines to be made and the group containing the paranoias has very sharp dissimilarities, not only clinically but etiologically and pathologically from dementia paralytica and its congeners. In the study of the different types of insanity the authors have made this very clear, however, and their characterizations of the different symptom groups is most admirably done.

The chapter on classification is philosophical and interesting. It analyzes the various systems now in vogue and sums up their essentials very judiciously. The arrangements of Ziehen and of Kraepelin are given first place, the former as the most logical psychological system, the latter as one of the most useful for present day students.

The major portion of the book is devoted to the description of the various insane symptom groups. These are sharply outlined and form a very decided addition to the descriptive literature of these very heterologous symptoms, making the work a very commendable one and a distinct addition to our psychiatric literature. JELLIFFE.

LEÇONS SUR LES MALADIES DU SYSTÈME NERVEUX. ANNEE 1897-1898. Par F. RAYMOND, Professeur de Clinique des Maladies nerveuses à la Faculté de Médécine de Paris, Médecin de la Salpêtrière.

This is the fourth volume of an annual series of clinical lectures delivered by Prof. Raymond at the Salpêtrière, collected and published by Dr. E. Ricklin.

The following is a list of the cases presented and discussed: Tumors of the Rolandic Area, Infantile Multiple Sclerosis, Alternate Paralysis, Polioencephalitis, Sclerosis, Double Ophthalmoplegia in Tabes, Asthenic Bulbar Palsy, Hemiplegia with Atrophy, Progressive Muscular Atrophy, Nosology of the Muscular Atrophies, Muscular Atrophy in a Tabetic, Tabes and Syringomyelia, Lead-Palsy and Syringomyelia, Case of Syphilitic Polyneuritis or Mercurial Polyneuritis, Case of Polyneuritis or Poliomyelitis, Traumatic Hysteria, Hallucinations of the Stump after Amputation, Case of Juvenile General Paralysis of the Insane or Central Syphilis, Myoclonia, Myxedema.

The lectures open with the history and results of the physical examination of the patient. The differential diagnosis then receives minute attention, each symptom being carefully weighed and then carefully interpreted.

The diagnosis having been made, reference is made to the literature and the prevailing ideas held by other well known observers. Prognosis and treatment then complete the lecture.

As these are clinical studies only without autopsical confirmation of the diagnosis, their chief value is to the student and the teacher.

To the latter, however, because of the great clearness of presentation, the ease of diction, the masterly disentanglement of complicating and confusing symptoms and the sound clinical sense and judgment displayed, these lessons should serve as a model and a guide.

J. RAMSEY HUNT (New York).

LES TICS ET LEUR TRAITEMENT. By HENRY MEIGE et E. FEINDEL. Preface by M. le Dr. P. Brissaud. Masson et Cie, Paris, France. G. E. Stechert, New York.

The senior author particularly has been interested in the study of tics and allied convulsive disorders, and has published a long series of noteworthy contributions during the past ten years. The work, which is some 633 pages, comes therefore with an authority of long and careful observation.

In the introductory preface Brissaud speaks of the desirability of recognizing the word *tic* as a most useful one, and dwells on the greater precision in meaning that it has acquired by reason of the authors' presentation. Recognizing the large rôle of the psychological element in the genesis of tics the authors open their discussion with a lengthy chapter on the "confidances of a tiquer," giving in great detail the entire mental history of an educated and well trained observer who suffered from a gradually developing "tic." "Son of a tiquer, brother of a tiquer, father of a tiquer, himself a tiquer, M. O... is the prototype of a tiquer." Such is the striking opening of this unraveling of a mental life of a patient bearing particularly on the subject of his affection. The history gives a characteristic summary of the entire clinical picture.

Following this chapter the authors present a historical critique summarizing most fully the gradual development of the neurological trend in the study of this condition.

In their pathogenic study special emphasis is laid on the differences in types of muscular contractions and the delimitations of the words spasm, tic, convulsive and spasmodic are carefully drawn. They follow the usage of Charcot and Brissaud in applying the word "spasm" to a motor reaction due to a pathological irritation at some point in the bulbo-spinal arc, to be a "tic," it is necessary that the cerebral cortex shall have participated. In further differentiation those motor reactions, inclusive with tics, in which the cortex is involved, the degree of coördination and of purposeful action is a most important element. Thus they say that the tic movement is a coördinated one, it is further systematic and finally it has a definite object or genetically has had at one time such an object.

Tics then are considered as tempestuous functional movements representing an act commanded by an exaggerated desire, and in which the resultant satisfaction of the movement may be still present or may have lost all significance, the impulsive habit alone remaining as a parasitic function as it were.

Successive chapters are on the Mental State, Etiology, Pathological Anatomy, on which subject the authors can offer nothing save their thought that there is an acquired lesion, based on some congenital anomaly, arrest or vice of development of the cortical association tracts, or of their infracortical association fibers, perhaps minute modifications not now recognizable in the present unsatisfactory state of technic.

Further chapters are on Studies on Motor Reactions, Accessory Symptoms, Tics of Special Muscle Groups, Tics of Writing, Tics of Speech, Evolution of Tics. An interesting chapter is on Gestures and Antagonistic Stratagems, a subject rarely thought of by many observers. Diagnosis, Distinctive Features, and Treatment are the concluding chapters.

The work is truly a most interesting and important one and destined to introduce a greater definiteness into our conceptions of the convulsive disorders in general. It is of the best style of the French writers, clear cut and practical, and merits a wide recognition.

JELLIFFE.

# News and Notes

**Docent Dr. H. Schlesinger** has been appointed Professor at Vienna.

**Prof. von Krafft Ebing** has recently died at his home in Graz of chronic nephritis.

**Dr. Alexander Pilcz** has been appointed privat docent at the University of Vienna.

**Dr. G. B. Pellizi** has received the appointment of privat docent in the University of Modena.

**Columbia University** will inaugurate a summer course in medicine in 1903. Drs. Pearce Bailey and R. Cunningham will be instructors in neurology.

**Review of Neurology and Psychiatry** is the title of a new monthly Review editted by Dr. Alexander Bruce of Edinburgh. This Review is to provide in English a journal similar in character to the shorter Reviews and Centralblätter published in French, German and Italian. The editor says that the Review is in no way intended to trench upon the field already occupied by the various larger British and American journals of nervous and mental disease.

This first number is a very commendable one and augurs success to the enterprise. We presented a résumé of its original articles in last month's issue and will analyze the February issue in April.

**The Bulletin**, a quarterly medical review which has been published since 1892 by the American Medical Temperance Association, has been consolidated with the Journal of Inebriety. The latter journal, which first appeared in 1876 under the editorial care of Dr. T. D. Crothers of Hartford, Ct., was the first and is still the only medical periodical in the world devoted exclusively to the scientific study of the neuroses and psychosis of spirit and drug diseases. ("Der Alcoholismus" excepted. Ed.)

**American Medico-Psychological Association.**—In accordance with a resolution passed at the Richmond meeting in 1900, the American Medico-Psychological Association has become affiliated with the Congress of American Physicians and Surgeons, and is now a constituent member of that body. Under the by-laws of the Congress it is required to hold a meeting every third year in Washington. This being the year for such meeting, the council has instructed the secretary to announce that the Association will meet in Washington instead of Providence. The dates for the Congress are May 12, 13, and 14, and the Association will continue in session on the 15th. Members of the American Medico-Psychological Association will receive programs from the general Secretary and announcements from the Committee of Arrangements of the Congress. The profit and pleasure of the meeting will be very much enhanced by the new affiliation. Sessions of this Association will be so arranged as not to conflict with the program for the general meeting. Some of the titles sent are as follows: Treatment of Morphine Habits by Hyoscine, by Dr. J. M. Buchanan; Report of a Case of Cerebral Lues, by Dr. J. E. Courtney; Dementia Præcox, by Dr. A. R. Defendorf; Report of a Case: Was He a Paranoiac? By Dr. C. A. Drew; The Toxemic Basis of Certain Brain Diseases, by Dr. W. E. Dold; Some Observations on the Insane: Blood Pressure, by Dr. W. R. Dunton, Jr.; Blood Conditions of the Insane, by Dr. H. C. Eyman; Psychology of Epilepsy, by Dr. Everett Flood;

Delusions, Hallucinations, and Illusions of the Pudic Nerve, Cerebrospinal Areas, by Dr. C. H. Hughes; An Epidemic of Typhoid Fever Due to Impure Ice, by Dr. R. H. Hutchings; The Physiological Demands in Hospital Food Supply, by Dr. W. H. Kidder; Insanity in the Negro, by Dr. J. F. Miller; Paranoid Dementia, by Dr. C. W. Page; Drill for Patients, by Dr. G. A. Smith; How Dr. Brigham Met the Challenge to Diagnose Insanity at Sight, by Dr. Stephen Smith; Pathology of Acute Delirium, by Dr. H. A. Tomlinson; The Paranoic Forms of Manic-depressive Insanity, by Aug. Hoch, M.D.

A symposium on the subject of Trauma and Insanity is contemplated. Papers are also promised by Dr. H. E. Allison, Dr. G. A. Blumer, Dr. A. E. Brownrigg, Dr. H. C. Baldwin, Dr. L. L. Bryant, Dr. Edward Cowles, Dr. J. B. Chapin, Dr. E. P. Chagnon, Dr. Edward French, Dr. G. H. Hill, Dr. Robert B. Lamb, Dr. P. L. Murphy, Dr. I. H. Neff, Dr. A. B. Richardson, Dr. B. T. Sanborn, Dr. W. P. Spratling, Dr. G. G. Wagner.

Dr. F. M. Powell of Glenwood, Iowa, has been reappointed superintendent of the Institute for Feeble-minded Children.

Dr. William Mabon, formerly of Ogdensburg, took up his new duties as superintendent of Bellevue, Fordham and Gouverneur Hospitals, January 1, 1903.

The last annual report of the London County Council shows that the increase in insanity for the year was greater than any other year excepting 1896. There was a total population of 22,155 under the care of the asylum committee January 1, 1902. The new asylum opened March last for the accommodation of two thousand patients is already filled, in part by transfer from other overcrowded asylums. The committee has already under consideration the necessity of proceeding with the construction of another building in order to provide for the estimated increase of five hundred *per annum*. At the present rate a population of half a million insane will be under care by the end of the present century. The chief causes alleged in the yearly etiological tables are hereditarv predisposition 27 per cent., and alcoholic excesses 17 per cent., both of which are thought to be much greater were the taking of more accurate data possible.

Dr. Liebeault, of Nancy, returned to the town of Favières where he was born seventy-nine years ago, to spend his birthday. His friends seized the occasion to place a commemorative tablet on the house where he was born. It states that A. A. Liebeault opened a new era in the medical sciences by his discovery of the systematic application of suggestion and induced sleep in the treatment of insomnia. He is the founder and leader of the so-called Nancy school. The inscription is signed by Voisin, Berillion and other members of the Paris Société d'Hypnotisme et de Psychologie, and by leading psycho-therapeutists of Germany, Sweden, Holland, and also by Vogt and Forel of the *Zeitschrift für Hypnotismus*.

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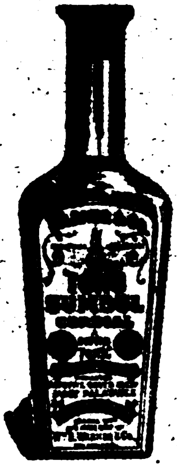

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The Official Organ of

The American Neurological Association
The New York Neurological Society
The Philadelphia Neurological Society and
The Chicago Neurological Society

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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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## Original Articles.

### REVERSALS OF HABITUAL MOTIONS, BACKWARD PRONUNCIATION OF WORDS, LIP WHISPERING OF THE INSANE, SUDDEN FAILURES OF VOLITION, REPETITION IMPULSES.[1]

By S. Weir Mitchell, M.D., L.L.D., Edin., of Philadelphia.

The present paper contains brief records of several forms of mental disorder. Some of these are too rare to be of much value, but are not without interest; others are unusual examples of well known abnormal conditions. That which I shall first describe as a very uncommon symptom of a diseased brain I do not find elsewhere mentioned. I am a little in doubt how to label the symptom. I must content myself with calling the phenomena Reversals.

These assumed two forms in the first case seen by me. The opposite of the thing willed was done, or else what it was meant to do was done, in a way which reversed the usual manner of doing it. The patient, a man in mid life, was an officer of the navy, of healthy descent, and unbroken health. Mentally he was very competent, but was what I should call anxious-minded, a man who put a needless amount of work into all he did. He first suffered after a time of unusual strain during the war of the rebellion. No warnings preceded the primary symptoms. He described himself as at this time exhausted in mind and body. When on the landing of a staircase intending to go up the next flight of stairs, instead of doing so, he found himself going down the flight he had just ascended, and going down backward. After descending a few steps he had the sense of mental shock, which all wholesome-minded men would experience under such conditions, and went up the stairs as usual although with some incomprehensible difficulty. Thereafter, at long intervals, and when overworked, this reversal happened. In the street if the action of walking suddenly ceased to be automatic,and he recalled the fact that he was walking, he would sometimes walk backward a few steps, and finally recovering himself, would go on as before. After a while he had great difficulty in ascending the stairs in the usual way, and was practically unable without effort to go up stairs foremost. When he was alone he was apt to go up backward, and generally, if not watched, descended the stairs in the same way. By and by this peculiarity began to be troublesome. He would go to a door to unlock it and would find himself making an effort to lock it. I had at one time notes of this interesting case, and have mislaid them. I do not think the peculiarity went much farther, and I believe he continued to be a man of mental value in all the ordinary affairs of life. I may add that during the years of this peculiarity he served in the war with distinction.

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[1] Read before the Philadelphia Neurological Society, Dec. 23, 1902.

Another curious case of the same kind was a middle-aged lady who had the not rare habit of turning to look at the end of a book before she began to read it in the usual way. At length, as she assured me, the habit became so fixed that she was positively unable to read a book in the normal manner until she had read a few pages at the end. Upon discovering that she was unable to resist the temptation, and that to continue to go backward through the book was hard to resist, she became greatly alarmed. She made earnest efforts to conquer, but I think never entirely overcame this curious difficulty, which she characterized as "the child of an over-indulged habit of curiosity to know how the book ended." Persistent effort to read in the usual way caused flushes and slight mental confusion, to which she usually yielded so far as to obtain relief.

Another case of still greater interest was that of a woman about fifty years of age, who had arterial degeneration, insufficient renal excretions, and attacks of mental disorder. These attacks in the opinion of the physicians who saw her, were caused by uremic conditions. In this case there was at times for hours a tendency to reverse actions, to put on her drawers over the head, or the undershirt over the feet, to put the shoes on the hands, or the gloves on the feet.

These curious reversals of habitual acts occurred only during the period when she was not in normal relations with the world, and of them she had no after-remembrance. In the intervals, although her mind might be said to be enfeebled, she preserved the excellent memory of a very well read woman, and was interesting, and not otherwise mentally disordered. This case occurred some years ago and was seen by me only in consultation, so that I have no notes except those imperfect ones which my own memory preserves.

A somewhat similar case, which I saw in consultation, occurred later in this city. The reversals were not as perfect nor as interesting as the one which I have just described.

I have seen, however, of late, a more notable example, the details of which I am not at liberty to give in full. The patient was a distinguished officer worn out in service. There were slight traces of albumin in the urine, with very insufficient excretions. He had a variety of morbid mental conditions, sometimes emotional weakness, but rarely delusions; sometimes slight explosions of anger and always difficulty in walking from general feebleness of the legs. At any time if left alone he would put on his drawers over his head, or his night jacket over his feet, and although he had a slight paralysis of the left side, he constantly used his crippled left hand where it would be usual to employ the right. Thus, instead of holding his knife in his right hand, he held it in his left hand. In place of starting with his right foot to walk, he put out first his left foot. He put on his left shoe first and his right glove first, both unusual ways with him, and with most of us. His shoes he put on his hands and at times tried to put his gloves on his feet. When the bath was prepared he sometimes made an effort to get into it head down in place of stepping into it, and had to be watched.

Another curious and interesting case, affecting speech, seems to resemble those which I have so briefly described. It was seen by me some years ago in the person of a dentist. He was subject to periodic headaches of extreme intensity. They occupied the left side of the head and were apt to last for a day or two, and to result in distinct paresis of his right hand, and in disturbance of language unlike anything I have ever seen. At times after the headache reached great intensity he either talked what seemed to be a jargon, which no one could understand, or else exhibited the following peculiarity of language. It affected chiefly the nouns. It is common enough to hear aphasic patients say "house" for "barn," "dog" for "cat," "road" for "path." Closely related substantives or ideas are employed. This person, however, used words which seemed to be in meaning the reverse of what he meant. In place of saying he would remain in-doors, he would say, "I will remain *out* of doors," when he meant "in-doors." If he wanted to say, "It is a clear day," he would say exactly the opposite. On one occasion he was made very angry by the incapacity of his wife to understand that when he said, "I do *not* want whiskey and water for dinner," he meant he *did* want them. It was interesting to observe that when in this condition, and it usually came toward the close of an attack of headache, he was perfectly well aware of his difficulty, and often refrained for long periods from expressing himself rather than cause the self-annoyance such speech was sure to produce. His headaches increased in intensity until he occasionally fell into a semi-comatose condition, or had a brief period of delirium lasting for some hours. With change of climate and occupation he entirely recovered his health, and lost both his headaches and these singular reversals of intended statements.

I think that you will agree with me that it is exceedingly difficult with our present knowledge to explain some of these singularities. I may add that the headaches were usually caused by any extreme fatigue; were not gouty, or due to eye troubles, and first arose after an attack of malarial fever at Panama.

Still harder is it to theorize on the following case. It stands alone in my large experience, and was seen by me when I was a student of medicine. An accomplished and scholarly gentleman was very ill with what we should early recognize today as a tumor of the brain. It was so diagnosed late in the disease, and proved after death to be a large soft cancer of the left anterior lobe of the brain. This patient was for a long while in bed suffering great anguish, with usually a condition of stupor, and with now and then a few hours of apparent clearness, during which he spoke what appeared to be a sort of gibberish. I was asked to sit up with him several nights, and, as the case interested me very much, I began to observe that what he said often consisted of repetitions. Having nothing else to do, after a couple of nights I began to amuse myself by trying to put down on paper what he said. An often repeated sound struck me as being "dog ho." This was repeated over and over. Then he would say, "Tac-im tac-im," and with this was then evident a desire for something which his wife could not understand. A great list of things was named without avail, until at last she recalled that his great household pet was a very beautiful cat, and when this was brought he exhibited pleasure and seemed satisfied. Suddenly it occurred to me that he had said backward what he had wanted: that "tac-im" was "my cat." The moment this idea presented itself to me I began to look over some of the notes I had made of his talk and found other illustrations which satisfied me of the correctness of my interpretation. For instance, "dog, ho" was "Oh, God." There were many other words capable of being thus used, but a large part of his very rapid output of language I could not understand. I comprehended it well enough, however, to perceive that when he came to use words incapable of clear backward pronunciation he mumbled over them, but every now and then fell upon some word that could be rendered backward, like "God," or "dog," or "cat."

I am perfectly clear that this speech was of the nature I describe. When I related my little discovery to Dr. William Pepper, the father of the late provost, who was in attendance on the case, he seemed to think it impossible; but when my father, who was also consulted, heard about it, I was able to satisfy him that I was correct. Neither of them had ever seen or heard of such a case, and it stands alone today in my not small experience.

I desire, secondly, to speak of the habit many of the insane exhibit of moving their lips when engaged in thinking. They have no wish to speak, but either do whisper or make lip and tongue movements too feeble to be heard as speech. This is a common symptom in people who for years remain on the border-line of insanity, sometimes near it, at others across it. Uneducated persons are seen to move the lips when reading to themselves. I make mention of this fact only to point out that these acts are examples of what are not altogether abnormal movements.

In fact, all efforts to mentally repeat remembered series of words without audible speech cause movements of the speech organs, of which we are unconscious. To prove this Dr. Scripture, of Yale, uses a thin membrane covering one side of a small cup. This is placed back of the teeth, and so that the membrane is in contact with the tip of the tongue. On the side of the cup is an opening connected by a tube to an ordinary drumhead carrying a lever which plays on the blackened cylinder of a kymographion. The subject, who does not know what is expected to occur, is asked to sit still and not to think speech. The result is a nearly straight line on the moving cylinder. Then he is asked to repeat verse or prose mentally without making any sound. At once the recording needle becomes agitated, and an irregular line results. Evidently the tongue has represented in minute motions that which was being repeated without sound.

The line between habit and obsessions is very narrow, and disliking the term obsessions I prefer to describe that I now deal with as despotic habits. How far they are despotic depends on temperament, mental state and previous duration of the habit. I cannot class them as always neurasthenic symptoms. They may sometimes be a part of the many phenomena of asthenic mental states, but they may also exist in vast variety in the healthy, or vary in intensity with mental health. They may be today mere controllable habits, and at another time despotically beyond volitional rule.

Some of the minor and harmless obsessions arise out of too long indulgence in habits of thought or action, the origin of which seems far to find. Thus, many wholesome people yield, as we all know, at times to a tendency to tread on alternate stones in walking; others resolve that to go to a certain place shall exact so many steps and no more. There is some faint satisfaction when it comes out as pre-desired, and if it fail, some mild dissatisfaction. Perhaps most of you have felt these tendencies.

One should watch all such valueless habits, since they may in the end become masterful. Even in persons of great intellect these habitual ways may become despotic. The healthy, as I have said, find a certain comfort in yielding. The sensitive and emotional, or those who live in perilous nearness to the boundary of unsoundness of mind, are so moved by not giving way to a morbid habit, as to suffer until, by yielding, they obtain relief such as any one who has tic, or habit spasm, obtains by ceasing to inhibit the abnormal muscular acts.

When by long habit some mental process has been connected with a form of physical movement, to break the relation may interfere with an associated intellectual activity.

Here is a curious illustration. When I was a boy at the Academy, under a severe master, the classes stood for recitation. Very soon boys thus placed sway, the whole line moving rhythmically from right to left and back again. For some reason, or whim, the master ordered this habitual motion to cease, and threatened punishment for disobedience. He discovered however that obedience caused a certain amount of mental failure, and at last, confessing as much, ceased to interfere.

The greater number of the oddest of these despotic acts seem traceable to no such innocent habit and are as inexplicable as facial tic, and of no related or other value. It would seem, in fact, as if deliberate ingenuity must have been needed to invent some of these acts, so strange are they and so complex.

Many of them are in some way related to numbers, like that recently reported to you by Dr. John K. Mitchell, where a queer hostility to the number three existed.

Perhaps no case more curious can be found than that of Samuel Johnson, as told by Boswell. No doubt it has been often quoted.

He was a prodigious worker, had some irregular movements which were analogous to habit chorea, and was subject to attacks of profound melancholy. Of his obsessions Boswell speaks thus:

"About this time he was afflicted with a very severe return of the hypochondriac disorder which was ever lurking about him. He was so ill, as, notwithstanding his remarkable love of company, to be entirely averse to society, the most fatal symptom of that malady. Dr. Adams told me that, as an old friend, he was admitted to visit him, and that he found him in a deplorable state, sighing, groaning, talking to himself and restlessly walking from room to room. He then used this emphatical expression of the misery which he felt: 'I would consent to have a limb amputated to recover my spirits.'

"Talking to himself was, indeed, one of his singularities ever since I knew him. I was certain that he was frequently uttering pious ejaculations; for fragments of the Lord's Prayer have been distinctly heard.

"He had another peculiarity of which none of his friends ever ventured to ask an explanation. It appeared to me some superstitious habit, which he had contracted early, and from which he had never called upon his reason to disentangle him. This was his anxious care to go out or in at a door or passage, by a certain number of steps from a certain point, or at least so as that either his right or his left foot (I am not certain which) should constantly make the first active movement when he came close to the door or passage. Thus I conjecture; for I have, upon innumerable occasions, observed him suddenly stop, and then seem to count his steps with deep earnestness; and when he had neglected or gone wrong in this sort of magical movement I have seen him go back again, put himself in a proper posture to begin the ceremony, and, having gone through it, break from his abstraction, walk briskly on, and join his companion.

"A strange instance of something of this nature, even when on horseback, happened when he was in the Isle of Skye. Sir Joshua Reynolds has observed him to go a good way about, rather than cross a particular alley in Leicester Fields; but this Sir Joshua imputed to his having had some disagreeable recollection associated with it."

The horseback incident here above alluded to I found with difficulty in Boswell's second volume. Johnson was seen when on horseback to turn suddenly and ride back a short distance with no apparent object. He rejoined his friends without making any explanation. Boswell regarded it as only another instance of the peculiarity already described.

There is one very rare form of mental difficulty which I do not find recorded in the books. I hesitate to give it a name. It consists in a suddenly acquired incapacity to do, not a certain class of things, but one particular thing. I know of two very sane and very intelligent persons who are thus troubled; one, a lady, writes many letters. Once in a year or two she finds herself unable to answer some single, and by no means unusual, letter. If she persists, she misspells words, gets the wrong words; as another puts it, is "word-crazy." Then a day comes when she tranquilly writes the letter. A member of her family had a similar annoyance which beset him variously. Once in New York he found himself unable to pack his valise. He went away; returned the next day, again failed, and finally called up a hotel servant to do the simple thing he could not do. The family is not neurotic.

The obsessions of childhood appear so far to have escaped study. In my own home one brother, in later days an army officer of distinction, had, as a child, a great dread of feathers, and of all fluffy objects. A bunch of cotton, or a feather, laid on the lintel of an open door kept him an easy captive. He could not pass over it or by it. Another brother, who also served in the Civil War as Colonel, would not, when young, go out of doors without an umbrella. This lasted for some years from the age of ten. How it ended I do not recall.

The following case is briefly reported as an example of great variety of despotic ideas:

Mrs. S. S., of Kansas City: Family history negative, never robust as a child. Married at twenty years. Tertian malaria at twenty-four years. At twenty-six years second child was born. Very severe labor and great shock to her nerves. A few months after, while sitting with the baby asleep in her lap one night, she was suddenly seized with a desire to cut the child's throat. She bolted the door of the room in alarm, lest she should get the knife which was in the next room. From this time for two years she was continually tortured by strong impulses to kill the child. At times she had suicidal thoughts. The same impulses returned to her after birth of third child, having been left alone with the child one evening. This time the impulse remained until her fourth pregnancy was established, when it left, not to return until several weeks after her delivery. This time it was brought on by two telegrams announcing her brother's death. Prior to this time she never had the homicidal feeling when with a third person. Now she had it all the time. Her age at this date was twenty-seven.

Her impulse to kill her children and herself remained up to her fiftieth year, although it became very much less and she had long intermissions of normal mentality. She became very nervous as years went by, and twice resorted to a sanitarium. While she was in a health resort, and at fifty years of. age, her obsessions changed absolutely. She developed the despotic need when doing minor acts to repeat them with unchanging numerical relation to the person she happened to think of at the time. Thus she lifts a book and must do it three times for a cousin, six for her husband, nine for some one else.

I give herewith the repetition and other impulses in the order of their development.

(1) She looked frequently in her water-pitcher, because if she did not there would be a snake in it. (2) She had to leave the house in "certain way" or else she could not "deliver her mind of the impulse," and must needs do it over and over again until she did it to her liking. (3) When making a call she would have to move a table or chair a few inches before she left. (4) While walking she had to step either *on* each crack in the pavement, or else over each crack. (5) She began to repeat very many actions in every day life six times (once each for father, mother, two sisters, one brother, and herself). This would apply to things like taking off shoes, combing hair, putting on articles of clothing, carrying a glass to her mouth, etc. (6) While out driving she would have to count six windows in each house. This, after a few weeks, began to reduplicate in multiples of six (*i.e.*, 12, 18 and even 24). She thought if she failed in this she would kill some one or else commit suicide. (7) On turning off gas she would have to repeat the operation again and again to be sure it was entirely turned off. (8) Seeing a light under the door one night while in bed she had to get up and rub the door sill with her hand, touching each finger evenly, eighteen times. If she failed she would not sleep. (This obsession only happened once.) (9) In a large closet in her room she must pass her hand all around the wall back of the clothes before she could go to sleep. (10) If there was the least wrinkle in the spread on her bed she could not sleep. (11) She was afraid to look at a locomotive for fear she would throw herself in front of it. (12) She was afraid to leave her seat in the car for fear she would throw herself from

the train. (13) She had to brush off her bed every night in order to get off any snake which might be on it. (14) Must brush off bed in order to smooth clothes.

The patient makes this statement in some notes she has made on her case:

At times, as I pass a door I am startled because I can almost see a person vanishing, although I know that no one is there, and I am almost afraid to look that way again because a dead person is so plainly in my mind." . . . "Sometimes I can not go to the bathroom this feeling is so strong."

Her mind was throughout clear. She was an efficient mother, wife, and housekeeper, and a great reader. She says: "I am like two people. I watch one, who is queer and erratic. I am, I think, the other also and am conscious and ashamed of my second self."

She has twice been under the care of Dr. J. K. Mitchell, and twice recovered entirely, being today without trace of her former peculiarities.

# A CASE OF MULTIPLE FIBROMATA CONFINED TO THE INTERNAL PLANTAR NERVE.[1]

By Wm. J. Taylor, M.D., and Wm. G. Spiller, M.D.

## REMARKS BY DR. WM. J. TAYLOR.

The case which I now report is to me unique and many of its features are of such interest that it seems worthy to be placed on record. I first saw this patient, then a young woman of twenty-seven and a mill-hand, in the autumn of 1888. Her mother was living and well, her father had died from accident, and she had five brothers and sisters all living and well. She had had smallpox when a child, but no other serious disease, and no specific history, nor has she ever had rheumatism. When about sixteen years of age she sprained her left ankle and since then it had always been weak and had a tendency to turn inward. In 1881, or seven years before she was seen by me, she began to suffer from pain and soreness in the sole of the left foot and inner side of the ankle. At first this was noticed only upon pressure, but gradually it grew worse until she could hardly stand upon the foot or sit with the foot in a dependent position. The pain at that time was not so severe on walking, but the foot was only free from pain when she was lying down. For two years before she was first seen, or in 1886, her work had been such as to require a good deal of standing, as she was working in a mill, and this seemed to aggravate the condition. For the past few months this pain had become more localized and was confined to spots along the inner side of the foot and ball of the great toe. After an attack of unusually severe pain the foot would swell and become red, and it would also swell at any time if she sat with the foot in a dependent position.

At this time, October 29, 1888, many small masses could be felt along the inner side and sole of the foot—these masses varied in size from that of a hickory nut to that of a small pea, and followed the distribution of the internal plantar nerve. They were movable, hard, intensely painful to the slightest touch, and some of them seemed to be immediately beneath the skin, while two small masses could be felt on the ball of the great toe; in all about ten small masses could be distinctly outlined. At times they became red and inflamed. They were so exquisitely sensitive that the slightest touch would give her agonizing pain—simply drawing the finger lightly across the skin of the sole of the foot would cause her almost to fall off the chair upon which she was sitting. The whole foot and particularly the sole of the foot sweated so, and particularly after any manipulation, that the sweat would stand out in beads all over the skin, and at times dropped to the floor.

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[1] Read before the Philadelphia Neurological Society, Jan. 27, 1903.

On November 1st, 1888, she was operated upon by Dr. William Hunt at the Orthopedic Hospital, where as Assistant Surgeon, I had the opportunity of helping him. Thirteen small tumors were removed from along the line of the distribution of the internal plantar nerve. These masses varied in size from that of a hickory nut down to a very small pea. They were shelled out from beneath the skin with the greatest ease, having very slight attachments. They were quite firm and hard in consistency and contained no fluid when cut open, but showed simply a white and glistening surface; one or two of the larger ones seemed to be composed of three or four small ones together. Following this operation she had quite a period of comfort, but at no time was she entirely free from pain.

In 1890 she was readmitted to the hospital under my care, stating that she had been much better for about a year after the first operation, but the pain had returned and was quite as severe as ever. I now operated and removed two tumors, one at the instep and the other at the ball of the great toe. Following this she had a period of comfort, but was readmitted in 1897 when I again operated and removed six tumors of the same general character and from the same region, one the size of a marble from in front of the tendo Achillis, and above the level of the ankle joint; again she was relieved. These tumors were pronounced to be pseudo-neuromata by Dr. Guiteras. All manner of medication was tried—iodine, tonics, iodide of potassium, different types of apparatus, bandages—in fact everything possible that could be suggested, was experimented with. During this time the pain recurred, varying in intensity, but of such a character that she was obliged to give up her occupation, that of a mill-hand, as she could not stand on her feet. There seemed to be a reproduction of the small tumors which increased in number and grew in size until June, 1902, when she was again admitted to the hospital, stating that the pain was of such a character that she could not possibly stand it any longer. The foot was now intensely sensitive —four or five tumors could be distinctly outlined—the slightest touch even to the skin over one of these tumors caused intense pain and profuse sweating of the foot.

On June 13th, 1902, I operated, making a long incision over the posterior tibial nerve just above the internal malleolus, and extending it down along the distribution of the internal plantar nerve, quite eight inches in length. Five small tumors were found along the course of this nerve and were removed; one large tumor was found deep down in the sole of the foot beneath the dense fascia, and it was only distinctly located by its extreme sensitiveness. Even when she was under the influence of ether pressure would cause her to wince. This tumor was especially large, about the size of a small walnut, and was attached to the internal plantar nerve at several points by rather long bands apparently of nerve tissue. The internal plantar nerve was found greatly enlarged, larger than an ordinary lead pencil. In view of her long-standing pain, I exposed and removed about five inches of this greatly swollen nerve, to which were attached several of these tumors. The wound healed without the slightest difficulty and all sensation was gone. For the first time in these many years (14) she was free from pain and although all sensation over a certain area was absent, her joy was very great. Her general health improved to a wonderful degree, and since that time she has been perfectly well and able to resume her work.

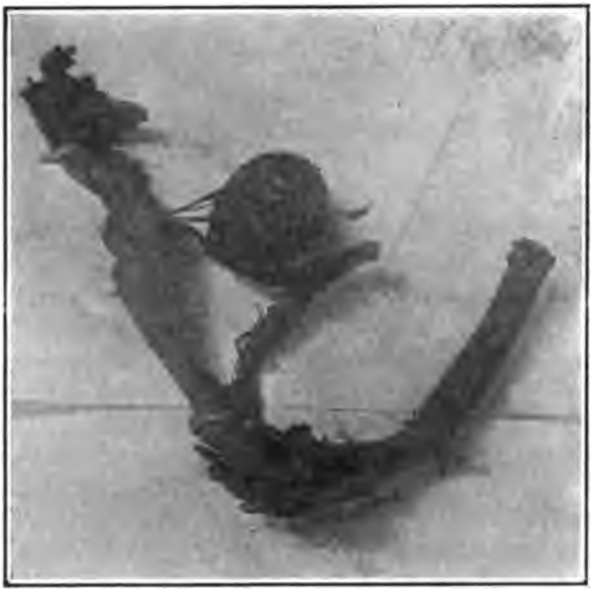
As I have stated, this case in my experience is absolutely unique, and Dr. Wm. G. Spiller, who has been kind enough to make microscopical examinations of the specimens, will report the neuropathological conditions.

November 29th, 1902, I examined the condition of the foot and found to be remarkably good. There is no pain whatever on walking, nor standing, nor has she at any time pain. The foot has been weak until very lately, but now it seems to have almost entirely regained its strength. Sensation is perfectly good in the skin of the sole of the foot, and there is tenderness at only one place in the whole line of the scar, and that is directly at the upper portion about on a level with the cut end of the nerve behind and above the malleolus. I imagine this sensitiveness must be simply at the end of the nerve, for I was not able to go entirely above the point of swelling in the nerve itself. The foot no longer sweats at all. She tells me that she is perfectly well, has great comfort and is able to work steadily.

### REMARKS BY DR. WM. G. SPILLER.

The report of a case of multiple fibromata confined to the ulnar nerve was published by Dr. W. W. Keen and myself in May, 1900. In that paper reference was made to the case of Dr. W. J. Taylor, now described at length. In view of the fact that Garrè, in speaking of his case, remarked that the presence of multiple fibromata in the skin of the sole of the foot was very extraordinary, and that v. Recklinghausen emphasized the immunity of this part and of the palm of the hand, it seems strange that in the two cases in which I have been concerned the hand was affected in one and the sole of the foot in the other. In one case the tumors were confined to the ulnar nerve, and in the other to the internal plantar. In Dr. Taylor's case a sprain of the left foot preceded the development of the symptoms of fibromata about four years, and the ankle after the injury remained weak. This accident may have been the immediate cause of the proliferation of the fibrous tissue in the left internal plantar nerve in a person predisposed to such proliferation. Dr. Keen's patient was a laborer, and trauma possibly aided in the formation of nerve tumors in the palm of the left hand. Trauma alone does not explain this singular process, or else multiple fibromata confined to a single nerve would be common, instead of being a very rare finding. Indeed, they seem to be more uncommon than the generalized neurofibromatosis. In the paper with Dr. Keen I referred also to a case of tumors confined to one nerve, reported by J. K. Mitchell and one by Bowlby.

In some cases heredity plays an important rôle, and members of different generations in the same family have exhibited neurofibromatosis. (Menke, Herczel, Bruns, Czerny.) It is impossible to believe that the process is purely an acquired one, and even in the cases in which the tumors have been confined to one nerve it is probable that the tendency to proliferation exists in other nerves, and that the proliferation may be called forth by trauma, or possibly by disease of the blood vessels. In the specimens sent to me by Dr. Taylor for examination the blood vessels removed with the nerve and tumors are greatly diseased, and the walls of the arteries are much thickened; some vessels indeed are thrombotic and without any lumen. This interference with the nutrition of the nerve may have aided in the production of fibrous tissue within the nerve bundles. The tumors are fibromata and contain only such nerve fibers as have not become degenerated by pressure; they therefore are not true neuromata in the sense that proliferation of true nerve tissue has occurred. In amputation neuromata there is a formation of young nerve fibers with the proliferation of fibrous tissue, so that these are truly neurofibromata, but in the specimens I have examined from Dr. Keen's patient and in those from Dr. Taylor's patient the fibrous tissue alone is proliferated.



A tumor dependent from the nerve trunk by several bands of fibers.

In one of the small tumors removed by Dr. Taylor which I have cut lengthwise and examined microscopically, medullated nerve fibers may be seen entering the tumor at one end, and then after entering separating and passing to the periphery of the tumor, and gradually disappearing from view, so that where they become widely separated from one another they can not be traced at all. As in Dr. Keen's case the proliferation has probably begun in the endoneurium of the nerve, and therefore the tumors are somewhat elongated, with their long axes parallel to the nerve. As the perineurium becomes thickened it offers some resistance to further growth of the tumor, and the proliferation of the fibrous tissue occurs more rapidly where resistance is less, viz., at the two ends of the tumor.

The tumors removed by Dr. Taylor, with one exception, are small, as were those removed by Dr. Keen from his patient.

It is easy to discover the cause of the intense pain in these cases. The nerve fibers are pressed upon and irritated by the proliferating connective tissue, and although many nerve fibers degenerate and thus lose the power of conducting painful stimuli, the process is progressive, and new nerve fibers are attacked as those previously diseased disappear.

One tumor, the size of a small walnut, removed by Dr. Taylor, is of peculiar interest, because it is dependent from the large nerve trunk by several thin bands of fibers. These seem on inspection to be bands of connective tissue, but one of these bands cut in microscopical sections consists of nerve fibers embedded in and surrounded by much fibrous tissue. This tumor has evidently grown from several small nerves, or else it has grown from one nerve and surrounded and embedded others within it.

The long duration of the process in Dr. Taylor's case and the repeated operations deserve mention, because removal of these fibrous proliferations of nerves has been regarded by some writers as liable to cause a change from fibrous into sarcomatous tissue. This has not occurred in Dr. Taylor's case.

I have discussed the subject of neurofibromatosis at length in the paper published in collaboration with Dr. Keen in May, 1900, and shortly after the appearance of this paper the excellent monograph on the same subject by Alexis Thomson was published. It is unnecessary therefore to say anything further concerning the specimens from Dr. Taylor's case.

# A CASE OF MYASTHENIA GRAVIS COMPLICATED BY ANGIONEUROTIC EDEMA.*

By Theodore Diller, M.D., of Pittsburg.

VISITING PHYSICIAN TO THE INSANE DEPARTMENT OF ST. FRANCIS HOSPITAL; NEUROLOGIST TO THE ALLEGHENY GENERAL HOSPITAL.

Myasthenia gravis or asthenic bulbar paralysis is doubtless a rare disease or symptom-complex. Campbell and Bramwell[1] in 1900 were able to collect only sixty cases from the literature; and of these but four cases, those of Collins, Berkley, Sinkler and Punton, were reported by Americans. Oppenheim[2] in an exhaustive monograph published in the same year gives extracts of all the cases reported up to that time. Appended to each of these monographs is an extensive bibliography. Although only two years have elapsed since Oppenheim wrote the preface to his monograph a considerable number of cases have been recorded in that time.

Among these are one by Freinberg[3], six by Frajersztajn[4], two by Buzzard[5], one by Buck[6], one by Batten and Fletcher[7], one by Frendenthal[8], one by Paul[9], one by K. Mendel[10], one by Guastoni e Lombi[11], one by Hall[12], one by Leifman[13], two by Burr and McCarthy[14], two by Jacoby[15], nine by Edwin Bramwell[16], two by Raymond[17], six by Goldflam[18]. The last named author expresses his conviction that the disease, or symptom-complex is of more frequent occurrence than true bulbar paralysis.

Thus it will be seen that 38 cases have been reported within the last two years, or more than half as many as have been collected by Oppenheim and Campbell and Bramwell in their monographs.

The cases reported by Burr and McCarthy, Jacoby, Paul and myself make a total published by Americans ten in number. Bramwell's personal observation of nine cases and Goldflam's detailed report of six cases would certainly seem to indicate that the disease is much less rare than it was formerly thought to be.

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*A paper read before the Philadelphia Neurological Society, October 28, 1902.

It is not now my intention to discuss the nature of this strange affection, or attempt an analysis of the reported cases, but merely to direct attention to the frequency of its occurrence, and to report briefly a case which is now under my observation. Although I have been able to find records of 38 additional cases reported since the publication of Oppenheim's monograph two years ago, no substantial addition to our knowledge of the disease it seems to me, has been made by their study; or at least enough to justify at this time a re-examination of the whole question as to the pathogenesis of the affection. Oppenheim's monograph may, therefore, be considered as a sufficiently adequate statement of our present knowledge of the disease.

The diagnostic criteria of myasthenia gravis are, according to Oppenheim, these: The combination of incomplete ophthalmoplegia externa with weakness of the hips and extremities; the step-like or slow development of paralytic symptoms affecting chiefly the muscles of mastication and the orbicularis palpebrarum, and the frequent involvement of the muscles of the neck; the pure motor character of the attack; the preponderance of myasthenia over paralysis in the affected muscles; the remittent course; the absence of true muscular atrophy with corresponding electric reactions of degeneration; the long duration of the disease; the frequent presence of myasthenic electric reaction.

My own case is briefly as follows: The patient is a woman aged twenty-nine years who has been married twelve years. She has borne three children, one of whom is dead; no miscarriages. Her mother died at forty-two of heat stroke; her father is living and well at fifty-seven. Three brothers and sisters are dead. No family history of nervous disease or tuberculosis.

The patient had diphtheria as a child, and scarlet fever since her marriage. She is a victim of alcoholism and was brought to the hospital in a condition of intoxication.

Two years ago she was compelled to take her bed and remain there during a period of five months on account of nervous prostration. During this attack she did not feel sick, nor did she suffer any pain. This "attack" is described rather indefinitely by the patient, but so far as I can gather it seems to have consisted of muscular weakness and general debility. She states that she was in bed because she was too weak to get up; that when she did so she became rapidly exhausted and was soon compelled to resume her bed again. Yet her symptoms did not seem to be neurasthenic in character. Her recovery from the so-called "attack" was not complete; for although she was able to be about most of the time since her period of five months in bed, she has been more or less tired out and exhausted.

On July 11 last she took a severe cold; and the same night was seized with a chill; and during the next twelve hours she suffered chilly sensations. At nine o'clock the next morning she found great difficulty in articulation; and her speech was well nigh unintelligible to others. This defective articulation has continued up to the present time, being, however, much more pronounced at one time than at another. She has never had any difficulty in swallowing or in mastication. Neither convulsions, mental disturbances nor unconsciousness occurred.

She was admitted to Dr. Griggs' service at St. Francis Hospital, July 14, 1902, with a temperature of 103.8° F. It gradually descended to normal within the next fifteen days, seldom going above 101° F. On July 31 it took a sharp turn upward and on each of the next three days touched 103° F. During the three following days it rapidly declined until it reached the normal, where it has since remained.

The rise of temperature on July 31 was coincident with the appearance of large circumscribed areas of edema located as follows: Summit of right shoulder, outer side of right upper arm, outer side of right elbow, outer and upper aspect of right forearm, outer crest of right ilium (this was the largest, its area being equal to that of the extended hand), two over outer aspect of right leg. These edematous swellings were circumscribed, non-inflammatory, and very painful. They came rapidly and disappeared rapidly, the period elapsing from the appearance of the first patch until the disappearance of the last patch having been about five days.

I saw the patient for the first time on August 7. Only slight traces of the edema were visible. The patient was free from pain. Mentally she was bright. The face and mucous membranes were pale. Her speech at once attracted attention. It was thick, slurring and indistinct. The face was mask-like; its muscular play during speech being much limited. The pupils responded normally to light and accommodation; the eyegrounds were normal; no ocular palsies were apparent. (The patient stated that for several days about the time of admission, she saw double.) Tested by resistance the facial musculature seemed weaker than normal. The patient chewed and swallowed without difficulty, but the movements seemed slow. The movement of the soft palate was weak and sluggish. The legs were distinctly weak, but she was able to stand. Her grasp of hands was less than normal—dyn.: R 40; L 30. No tremor; no atrophy.

Sensations for touch, pain and temperature were normal. No dermography. No pain on pressure. Knee and Achilles jerks were sluggish. Skin reflexes sluggish. Thoracic and abdominal organs seemed normal.

During the examination articulation was defective, becoming more and more so, and at the end was scarcely intelligible.

Upon admission the urine showed albumin and granular casts, but these were not discovered in several examinations made afterwards.

Blood examination: (Dr. Disque). August 10. R. B. C., 3,988,000 (average of 150 squares). August 24. R. B. C., 4,226,000 (average of 150 squares).

Ever since her admission to the hospital great variation in her power of articulation was noted. Speech was usually much clearer after a long period of rest. In general it was better in the morning and grew worse toward night.

August 12—Dr. Nealon notes speech is much worse tonight than at any time since admission. The muscles of mastication tested before and after a meal exhibited no difference in strength.

August 21—Patient complains that her hands are weak, that breaking a piece of bread tires them out.

September 23—General improvement has occurred. Articulation is much clearer. Her color is better. She is cheerful, free from pain. Appetite is good; bowels regular. She sleeps well. She expects to leave the hospital within a few days.

(A battery not being available, an electric test to determine the presence of myasthenic reaction could not be made.)

That the case just described is one of myasthenia gravis can, I think, hardly be doubted. The illness of two years ago must, I believe, be accounted as the beginning of the present illness. The diagnosis rests upon the long course of the disease, the weakness of the extremities, of the face and soft palate; the occurrence of diplopia; the absence of sensory and mental symptoms and of atrophy; but above all upon the peculiar speech defect and the fact that it varied greatly in degree, being always much more pronounced after conversation. The relation between angioneurotic edema and myasthenia gravis is a most interesting one; and one which I believe has not heretofore been recorded; and yet one which is difficult to determine since the nature of either affection is unknown. That both were due to the same underlying cause seems, however, probable, and to me a theory of vasomotor lesion seems inviting. Collins[19], from the study of a case reported by him, inclined to the view that the sympathetic was at fault because of the attacks of collapse and cyanosis from which the patient suffered. No other author seems to have offered this suggestion.

I am indebted to my colleague, Dr. Griggs, for the opportunity to study and report this case.

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# Society Proceedings

## NEW YORK NEUROLOGICAL SOCIETY.

December 2, 1902.

The President, Dr. Joseph Collins, in the chair.

*Generalized Scleroderma with Vascular Spasm of the Tongue.*—Dr. Joseph Fraenkel presented a girl showing generalized scleroderma and called attention to a peculiar symptom. After the use of the tongue the patient complained of some pain in this organ, and inspection showed a vasomotor spasm leading to the production of an area of whiteness and dryness on the tongue.

*A Case of Congenital Multiple Sclerosis.*—Dr. Fraenkel also presented a girl of seven years. The parents are cousins. Five other children are in the family, and are living and well. One child died in infancy, having apparently suffered from the same disorder as this patient. The child presented was born after an easy and natural labor, but did not walk and talk. The head was small and the eyes had the Mongolian set. Vision and hearing were apparently normal, and the child was docile. Nystagmus was observed on making the little one look upward. The patient was unable to stand or sit up without assistance, and the gait was typically ataxic. The speech was defective and somewhat syllabic. There was no actual motor paralysis. There was marked incoördination of the muscles of the upper extremities of the intentional type, more marked on the right side. The reflexes were normal. Both feet were very red in consequence of vasomotor disturbance. The only diagnosis that could be made was congenital multiple sclerosis.

*A Case of Subacute Poliomyelitis.*—Dr. J. Ramsey Hunt presented a man, thirty-nine years of age, a boiler-maker by occupation. In 1889 there was an initial lesion apparently followed by a mucous patch on the tongue, but he received no internal treatment. Nine years ago he worked for a time in a white lead factory, but developed at that time no symptoms of lead poisoning. His present trouble dated back to last January. The first symptom was a weakness or stiffness of the arms, but he was able to continue at his arduous work of wielding a sledge hammer. Finally, one day he became paralyzed. When seen, five months later, the muscles about the left shoulder were paralyzed, and gave a complete reaction of degeneration while the other muscles were normal. One month later some weakness was experienced on the other side, and this was followed by almost complete paralysis about a week later. There were no electrical changes, but there were fibrillary twitchings. The reaction of degeneration appeared in two or three weeks in those muscles corresponding to injury at Erb's point, but some improvement subsequently took place. The paralysis at the present time had the same localization. There was an exaggerated tendon reflex, but no sensory symptoms were present. The man was under large doses of iodide at the time of the second paralysis.

Dr. Pearce Bailey said that he had seen two similar cases, in which the course was acute and was localized in the shoulder girdle muscles. Both of these patients were syphilitic. He regarded the condition as a localized syphilitic myelitis, probably associated with hemorrhages.

Dr. B. Sachs said he was disposed to look upon this as a case of specific amyotrophic lateral sclerosis.

Dr. Fraenkel said that it was an old notion that lead paralysis developed in the muscles most used, and this seemed to apply to the case under discussion. He was not willing to entirely exclude lead.

Dr. Joseph Collins presented for Dr. I. Abrahamson a similar case, occurring in a man of forty-eight years, a plumber's helper. He had had syphilis when thirty-six years old, for which he had received treatment for two months. He was well until the last of August, 1902, when he noticed, while wielding a hammer with the left hand, that the left upper extremity was becoming powerless, and from that time to this he had not been able to use this extremity. Two or three weeks afterward he complained of weakness of the right forearm. A few weeks later he noticed that the left shoulder was shrunken. At present there were: (1) Atrophy of the left shoulder muscles, biceps, triceps and forearm muscles, especially the flexors and atrophy to a slighter degree of the flexors of the right forearm; (2) fibrillary twitching of the most atrophied muscles; (3) exaggeration of all the tendon jerks; (4) weakness of the legs; (5) pin-point pupils; (6) no sensory disturbances, and (7) no typical reaction of degeneration in the atrophying muscles. The case was considered to be one of amyotrophic lateral sclerosis on a syphilitic basis. It was not unlikely that the pathological process was a focal syphilitic degeneration of the cervical cord, the ventral horns being principally involved. The primary change was probably in the vessels. Clinically, this case, and the case presented by Dr. Hunt, must be looked upon as amyotrophic lateral sclerosis. Dr. Collins said he referred to this subject to a greater length in his paper about to be read.

Dr. Hunt said he believed his case differed from an amyotrophic lateral sclerosis in its acute onset and course. The onset must have been accompanied by a great deal of edema.

*Escape of Cerebrospinal Fluid Through the Nose.*—Dr. Adolf Meyer presented a young man, who in 1894 injured the vertex by diving into water and striking a rock. Since that time there had been increasing severe headache, and in 1897 ocular symptoms appeared. After a while both eyes became blind, the last sector from which vision disappeared being the right upper one of the field of vision. In 1899 he experienced peculiar attacks of numbness on the left side of the face, occurring from ten to fifteen times a day. In August, 1900, there had suddenly appeared an oozing of fluid from the right nostril. This was followed by an improvement in all the symptoms. At the present time there is no facial palsy, but on opening the mouth, the jaw deviates to the right. Several times the patient while suffering from a cold, causing occlusion of the nostrils, had gone into a deep sleep. Last May, without any such occlusion of the nose, there was a general convulsion, and these attacks were repeated this Fall. Some months ago there was a strong tendency to walk in a circle. The speaker said that he had seen the patient soon after the oozing had begun, and then it was possible to demonstrate in the fluid reducing substances. At present, this fluid contained glucose, so that it was undoubtedly cerebrospinal fluid.

Dr. William M. Leszynsky said that he had studied the case reported by Dr. W. Freudenthal. That patient was a lady of about fifty-five years of age, who suffered a good deal from headache. There was a mild optic neuritis affecting both eyes, and the fluid escaped from the nose periodically in large quantities. There was no somnolence, and the patient was chiefly disturbed by the discharge of fluid.

Dr. B. Sachs said that he had had under observation for four years a girl afflicted with a tumor of the brain, its presence having been demonstrated by trephining. The girl had improved very much, although blindness persisted. For more than a year before there had been periodic oozings from one nostril. Examination proved the fluid to be cerebrospinal. The symptoms had been greatly relieved by the oozing of this fluid.

Dr. J. Ramsey Hunt said that he had seen in Bellevue Hospital last winter a woman who had been shot through the head. There was paralysis of the motor portion of the fifth nerve and a partial paralysis of the seventh nerve on the left side, with electrical changes showing the peripheral origin. The patient recovered quickly, and left the hospital in two weeks. At this time there was an occasional and scanty oozing of perfectly clear fluid from the nostril. It probably resulted from the comminution of the ethmoid and frontal bones.

Dr. Meyer said it seemed to be absolutely certain that the optic chiasm had been destroyed in his case by a tumor.

*Amyotrophic Lateral Sclerosis.*—Dr. Joseph Collins presented a paper on this subject. He said that this was among the rarest of all organic nervous diseases, the diagnosis having only been made in this clinic seven times out of about seven thousand cases. In ten years at the City Hospital, where chronic nervous diseases were very common, he had not seen more than seven or eight cases. The general knowledge of the disease dated from Charcot's classical description in 1874. The following case was reported very briefly: The patient was a woman, thirty-seven years of age, who complained of pain in the head, and in the muscles supporting the head. There were atrophy of the muscles of the shoulder girdle, neck and hands, slight spasticity of the muscles of the upper extremities and Babinski's phenomenon. There were early and profound bulbar manifestations. The duration of the disease was about three years. This patient also suffered from what he took to be major hysteria. The chief pathological findings were: (1) Uniform disappearance of the ventral horn cells throughout the entire cord, affecting possibly the dorsal region more severely than the cord enlargements; (2) a zone of degeneration in the cervical and dorsal region encircling the horns; (3) a marginal strip of peculiar shape in the lumbar region; (4) deformity of the ventral face of the cord, due to a concave sinking in of the periphery; (5) a neuroglia proliferation in the degenerated white matter and also in the anterior horns at the cord enlargements; (6) evidence that the neuroglia proliferation was older in the cervical region; (7) atrophy of the anterior roots; (8) distinct evidence of cell degeneration. The pyramidal tracts were intact. The question of whether the peripheral motor neurones or the central motor neurones were the first to be involved could not be settled. It was probable that in some cases the peripheral, and in other cases, the central were first affected. He thought the lesion in some cases began in the anterior horn, and the destruction of the cells there resulted in part in the disease, and accounted for the pathological findings. It was the decay of the column cells which caused the degeneration of the white matter in the case just cited. Destruction of these column cells led to the destruction of the fundamental columns. It was true this did not occur in every case because in such cases the destructive lesion of the spinal cells confined itself to the root cells and the column cells were not implicated. This probably explained all those cases of progressive muscular atrophy with changes in the white matter. There were now eleven cases on record in which the pathological process involved both systems of neurones in their entirety. However, the disease might exist in its most typical form without any involvement whatever of the pyramidal tracts. The changes in the spinal cord sometimes extended to the posterior columns, though the significance of this was not yet known. The speaker thought it must be admitted that amyotrophic lateral sclerosis was a different disease from spinal progressive muscular atrophy, because of the different clinical course and anatomical changes. In amyotrophic lateral sclerosis there was a poison capable of destroying both the white and gray matter. Whether this disease was a part of primary progressive muscular atrophy was another question, and, in his opinion, should be answered in the negative. The etiology of amyotrophic lateral sclerosis was very obscure. After a careful examination of the entire literature he had selected the records of 94 typical cases, and had added to them 9 of his own, 4 of them with autopsy. Of the 103 cases, 54 were males and 49 females. It was generally held that the disease was more frequent between thirty and forty, but in the hundred selected cases the fourth and fifth decades were found to be equally liable to the affection. The average duration of the disease was two years, though the minimum was a few months, and the maximum nine years. The upper extremity was affected first in 39 cases; the lower in 14, and the upper and lower extremities simultaneously in 11 cases, while the disease came on with bulbar symptoms in 21 cases. In the last mentioned cases atrophy or spasticity or both appeared very soon in other parts of the body. Trauma did not seem to be an adequate cause of the disease. Overwork, and especially the work of gold-beaters, embroiderers, and others, calling for exhausting use of special sets of muscles, did not seem to enter into the etiology. The disease occurred most among the working classes. A number of cases had developed shortly after parturition. Six of the patients gave a history of syphilis, but this was probably not more than the actual percentage of syphilization. In two of these cases, both his own, there might have been some relationship between the syphilitic infection and the amyotrophic lateral sclerosis. It was worthy of note that many of these patients had had syphilis, and had been workers in lead.

Dr. B. Sachs said that he thought the term, amyotrophic lateral sclerosis, was a far better clinical than anatomical designation. Clinically, the disease was recognized by the combination of spastic paraplegia with atrophic paralysis. The clinician could hardly avoid being impressed with the fact that there was not a single anatomical lesion for all these cases. In recent years he had seen quite a number of cases which he had labeled "spinal syphilis of the amyotrophic type," but he would not think of classifying them as true amyotrophic lateral sclerosis. He believed the relationship between amyotrophic lateral sclerosis and progressive muscular atrophy was closer than Dr. Collins supposed. Some cases of progressive muscular atrophy went on for a very long time without lateral column symptoms; in other cases, spasticity was an early symptom. These differences seemed to be explicable by a difference in the acuteness of the disease as affecting the gray matter. In progressive muscular atrophy of the ordinary type the process was a very much slower one than in amyotrophic lateral sclerosis. He would not be willing to draw a sharp line of demarcation between the two diseases, for, he felt they were closely allied. He had been very much impressed with Gowers' lecture on abiotrophy or special vital defect. He doubted if those afflicted with either one of these two diseases would have developed them if they had not been born with some point of least resistance in the gray matter of the cord. He desired to call attention to the fact, that degeneration of the gray matter was not always followed by that degeneration of the white fibers which one would expect from the close association between the two which is assumed by the neurone theory.

Dr. J. Ramsey Hunt said that Dr. Spiller, of Philadelphia, had reported a case of amyotrophic lateral sclerosis of very acute onset. He found degenerations in the association tracts of the cortex, and suggested this was the anatomical basis for many of the mental symptoms not infrequently described.

Dr. Louis Faugères Bishop said he had at the present time in his hospital service a woman with well-marked amyotrophic sclerosis, and the husband with right hemiplegia. The latter developed last. He was disposed to think that both had a common cause, probably syphilis.

Dr. Collins, in closing, reiterated his belief that spinal progressive muscular atrophy and amyotrophic lateral sclerosis are different clinically, etiologically and pathologically. To him the special feature of interest was that in some cases there was a transitional stage in which the lesion was limited to the commissural cells binding together different portions of the cord. He did not see anything in abiotrophy having bearing upon amyotrophic lateral sclerosis. The latter was an acute, violent infection. Weak persons generally were spared, he thought, all through life from acute intoxications or infections. The etiological factor of the disease would probably be determined by the physiological chemist.

*Officers Elected*—Dr. Pearce Bailey, President; Dr. J. Arthur Booth, First Vice-President; Dr. Frederick Peterson, Second Vice-President; Dr. B. Onuf, Recording Secretary; Dr. F. K. Hallock, Corresponding Secretary and Dr. G. M. Hammond, Treasurer.

# PHILADELPHIA NEUROLOGICAL SOCIETY.

December 23, 1902.

The President, Dr. John K. Mitchell, in the chair.

Dr. S. Weir Mitchell read a paper on Obsessions (see p. 193).

Dr. Charles K. Mills remarked that one of the most interesting points in Dr. Mitchell's communication was the fact that in the cases reported the individuals had been not only of fair, but in some cases of high intellectual attainments. He had himself seen many cases of obsessions, fixed ideas or imperative conceptions more or less expressed in action, but most of these were in those who were either suffering from or tending toward some form of mental disorder.

A very important point is the presence of these obsessions in the early stages of dementia præcox. Dr. Mills looked with great anxiety on the case of a young person in whom this tendency to obsessions of any sort is prominent and persistent. He had followed several such cases for a number of years, and had seen them terminate in some form of dementia præcox.

Dr. Francis X. Dercum said that to his mind the question of obsessions was associated with the conception of a neuropathic state. While it is perfectly true that many of these individuals appear to be otherwise normal, we must remember that in a large number of cases the patient is in a transitional stage, and that we are dealing with a psychosis which will go on to later development.

There is a neuropathic basis in all of these cases. In neuropathic cases, fatigue may be expressed differently from what it is in ordinary individuals.

*A Case of Paresis in Which a Remission Had Lasted Four Years.*—Dr. Wm. Pickett reported this case. The man could not be shown. He had been conducting a bakery for the past four years, though his pupils are strikingly unequal, and are absolutely immobile, and his knee-jerks are absent. The mental symptoms of classic paresis appeared in the summer of 1898 and faded again in December of that year.

Dr. Pickett also reported a case of circular paresis and one of paranoia with double personality.

Dr. Charles K. Mills, referring to the organic basis of hallucinations and delusions, mentioned a case which he had seen some years ago. It was that of a man of great intelligence who had had an apoplectic stroke, the symptoms indicating that the hemorrhage had been about the oblongata and oblongata spinal transition. The symptoms were persistent hiccough and impaired sensation, muscular and cutaneous, with some involvement of the cranial nerves. Among other symptoms which persisted was the delusion that he had his exact counterpart in bed alongside of him. He afterward made a partial recovery. The speaker believed that, at least in some cases, hallucinations do have a local organic basis.

Dr. Francis X. Dercum remarked that cases of the circular form of paresis as illustrated by Dr. Pickett's patient were exceedingly rare. He had seen only one case. He considered as well taken the point made by Dr. Pickett that cases with a neurasthenic stage should not be regarded as cases of circular paresis. Cases of paresis are often seen in which there is a hypochondriacal condition before the expansive stage comes on.

Dr. William G. Spiller said with reference to hallucinations with organic lesions that he had seen a case in which word-deafness was a persistent symptom. There was also mental failure with auditory hallucinations. A cyst was found in the temporal lobe on the left side.

*Amyotrophic Lateral Sclerosis, with a History of Acute Poliomyelitis in Childhood.*—This case was presented by Dr. Alfred Gordon. The patient was a boy fifteen years of age. At the age of one year he had infantile palsy affecting the right arm and left leg. Eight years later he had a series of infectious diseases. Later he had two fractures of the right arm. This was followed by symptoms of amyotrophic lateral sclerosis with atrophy of the scapulo-humeral type. In the lower limbs the muscles are contractured and markedly wasted. In the upper, the muscles on the right are almost completely wasted and on the left to a great extent. Some of the atrophied muscles present also increased mechanical irritability and fibrillary contractions. The muscles show either a marked diminution of electrical irritability or complete loss. Some muscles present R.D. The atrophy affects also the muscles of the thorax. Some of the deep reflexes are lost and some exaggerated. The latter is particularly marked for the patellar tendons on either side. As a matter of coincidence, the patient's thyroid gland is enlarged.

*A Case of Tumor of the Brain.*—This was reported by Drs. Charles W. Burr and Ralph S. Lavinson. The patient was a lady fifty-five years of age, who five years ago had a cancer of the left breast removed. There was recurrence. Slight muscular twitching of the left arm and hand was noticed with some loss of power in the hand and leg. Later she had attacks in which there was a continuous tonic spasm of the right arm lasting for hours at a time.

At the autopsy a small growth in the middle part of the ascending frontal convolution on the right side was found.

*Double Consciousness or Amnesia.*—This paper was read by Dr. F. Savary Pearce. The patient, a young man, had been in perfect health until August, 1902, at which time he was in a trolley accident, and was struck on the head, and was unconscious for a moment. He was apparently well afterwards and went about his business, that of a grocer. On October 10th he left home, and that was the last seen of him for ten days. His team was brought home by two boys to whom he had given it. When he "came to himself" he was near Baltimore. He then turned and walked to his home in Philadelphia. After his return he slept almost continuously for three days. He then apparently recovered, excepting the entire loss of memory of events of three years past. His physical condition is now normal. His memory for recent events is good, but he remembers nothing from September, 1899, up to the time that he "came to himself" near Baltimore. No evidence of hysteria could be made out. The eye-grounds and the fields of vision are negative. X-ray examination of the skull is negative, and there are no evidences of fracture, nor signs of palsy anywhere.

The possibility of existence of minute diffused hemorrhages in the cortex of the cerebrum, or of *commotio cerebri* alone, with disassociation of function of the cerebral neurones, particularly those of the frontal lobes; or the functional disturbance of the cells in which the latest acquired conceptions and perceptions are resident, were given as possible explanations of the rare affection.

Dr. D. J. McCarthy suggested that this might be a case of epileptic ambulatory automatism. The traumatism with these vague symptoms makes it possible that it may be classed under this head. Such patients have been known to walk hundreds of miles.

The President suggested that this might be a case of post-epileptic loss of consciousness. He referred to a patient in this condition who kept a diary for several weeks, and who after coming to himself had no recollection of the events which he had recorded, though it was proved from other sources that the statements of the diary were correct during the above period.

*A Case of Gumma of the Brain with Operation.*—This case was reported by Dr. T. H. Weisenburg. The patient was thirty-seven years of age with a history of syphilis. Five years ago he fell on his head and was unconscious twenty-four hours. Eighteen months ago he again struck his head. He had headache, vomiting and convulsions beginning in the right hand. In June, 1902, Dr. John B. Roberts operated, and found a gumma of the dura and of the brain. The patient made a good recovery, but the convulsions still occurred. An interesting point is that there has been a return of muscular power although about an inch of the motor cortex has been removed.

# CHICAGO NEUROLOGICAL SOCIETY.

Joint Meeting with the Chicago Medical Society, Dec. 10, 1902.

The President, Dr. Wm. A. Evans, in the chair.

*The Changes in the Spinal Cord and Medulla in Pernicious Anemia.*—This was the title of an address delivered by Dr. Frank Billings.

Causes.—Among other things, Dr. Billings said that our knowledge of the cause of pernicious anemia is not satisfactory. We recognize certain contributory factors and infer a condition of toxemia which has not been proved. The disease so frequently follows the infections, like syphilis, malaria, typhoid, la grippe, *etc.*, that it is impossible to ascribe any specific relation to them. The disease occurs more frequently in women than in men. It may occur at any age, but it is more common in the fourth decade than at any other time in life. Intestinal parasites have been found present in the disease, especially the *Bothriocephalus latus* and the *Ankylostoma duodenale.*

The condition of profound anemia, with deformed red cells, the presence of fetal red cells in the circulating blood, the presence of an abnormal amount of iron in the liver, together with degenerative changes in the muscles and in the marrow of bone, is a reasonable hypothesis for the assumption of the presence of a circulating poison—a hemolytic toxin—as the fundamental cause of the disease. The source of the poison has been the subject of many theories.

It is not improbable from the evidence we have that pernicious anemia is due to some hemolytic toxin, but whether of bacterial or autogenetic origin we cannot at the present time definitely say.

Symptoms.—The symptoms of pernicious anemia are chiefly those due to a profound anemia manifested by weakness, lessened endurance, with dizziness, dyspnea, palpitation of the heart, *etc.* In probably a large percentage of the cases the body weight is preserved. There is a tendency to subcutaneous, submucous, retinal and other hemorrhages. Gastro-intestinal disturbance is very commonly present, and especially diarrhea. The appetite is often poor or capricious. The disease is associated with nervous phenomena in the great majority of cases; usually, however, subjective in character. The spinal cord lesions, which are now recognized as occurring in a small percentage of the cases, may appear as one of the earliest manifestations of the disease, or they may occur late, and may be manifested slightly or not at all up to the time of death.

Dr. Billings gave an analysis of forty-one cases of pernicious anemia that he had seen during the last few years. The consensus of opinion is that the nervous lesions are due to a toxic agent, which is also responsible for the anemia when it is present. The fact that the lesion does not involve, as a rule, a whole neurone or system; that the trophic cell is usually not disturbed in either the cortex, the spinal gray matter or the posterior ganglion, and that the ultimate primary change is one of degeneration of the nerve fiber itself, speaks for the effect of some blood-circulating toxin. The fact, too, that the brunt of the lesion occurs in that part of the tracts involved farthest removed from the trophic center, where the nerve fiber is the least protected by its nutritive cell, is evidence of a toxic cause.

Conclusions.—The conclusions which one may draw from a study of the subject are: (1) That there is a well-established relation of diffuse cord degeneration with pernicious anemia; (2) It seems probable that the hemolysis and the cord changes are due to the same toxin; (3) While the source of the toxin is unknown, the fact that gastro-intestinal disturbance is so common in the disease would lead one to suppose that it is of intestinal origin; (4) The diffuse degenerations of the spinal cord which occur in conditions without pernicious anemia do not appear to differ essentially from those of pernicious anemia; (5) It is possible that a common blood-circulating poison exists which may expend its force on the blood in one individual, on the nervous apparatus in another, and coincidently on the blood and spinal cord in others.

Dr. Sydney Kuh said that Dr. Billings, in speaking of the etiology of pernicious anemia, quoted the statistics of some foreign authorities based upon 270 cases of pernicious anemia. In these statistics the statement was made that of this number of patients 22.4 per cent. had suffered from syphilis, and from this the conclusion was drawn that syphilis is a factor in the etiology of pernicious anemia, although not a very important one. This statement Dr. Kuh believed was misleading. Erb and Kuhn, and he himself, had tried to find how frequent syphilis is among patients in general, and it is quite remarkable to see how exactly the statistics of all three agree, the conclusion being that 22 and 23 per cent. of patients have been infected with syphilis.

There is no doubt that the toxin theory of the origin of pernicious anemia is by far the most plausible one. A variety of diseases, as atheroma, pellagra, *etc.*, can produce lesions in the spinal cord exactly similar to those mentioned by Dr. Billings. Dr. Kuh had seen one case in which the changes in degree and extent corresponded very closely to what had been described and demonstrated by Dr. Billings. In this patient, who had syphilis of the spinal cord, the parenchymatous and interstitial changes seemed absolutely independent of the distinctly syphilitic changes, and he attributed them to the syphilitic toxins.

Dr. Maximilian Herzog thought that it was not absolutely necessary to attribute the changes in the cord to toxins, because secondary anemia never leads to such changes. Secondary anemias are never as severe as initial or primary anemias. Secondary anemias do not kill the patient; he dies from tuberculosis or from some other condition, but not from the anemia itself. Dr. Herzog had never seen a secondary anemia like a primary anemia, with a blood count of 300,000. He thought malnutrition was more responsible for the changes in the cord than the toxins.

Dr. E. R. Moras stated that from observation and reading, and from cases he had seen, he had come to the conclusion that the fundamental cause of pernicious anemia is a chemic poison which exists in a relative quantity in the blood of probably every human individual or animal. The absolute quantity in each individual is determined by the habits, environment and special occupation and diet. Chemical analysis demonstrates that the acid produced in those conditions which are analogous to those found in the blood and tissues of pernicious anemic patients, is formic acid.

Dr. Archibald Church stated that about nine months ago he had under observation five cases of pernicious anemia with nervous manifestations. He had notes of about eighteen cases. Three years ago he called attention to the fact that in some instances spastic conditions were present, and that these very frequently terminated in flaccid paralysis. As the disease progresses it takes on the type of an ascending myelitis. Paresthesia is sometimes followed by anesthesia. In at least two cases he had seen a band of paresthesia or hyperesthesia followed by anesthesia gradually approach the trunk and chest, reach the neck, and terminate in Landry's paralysis. He was convinced that in some instances the gray matter is seriously involved. Recently he had followed to a fatal termination a case in which the lower limbs were at first in a spastic condition, and subsequently became flaccid and finally paralyzed; bed-sores developed, and the muscles of the trunk showed degeneration. Later the muscles of the hand and forearm showed decided atrophy, with fibrillary degeneration.

A point of some importance in the clinical onset of this disorder is that the mental picture is somewhat peculiar, owing to the probable impoverishment of the nutrition of the brain. These patients often present eccentricities of character and temper; they have hallucinations of sight; very commonly they will misinterpret things; they see objects crawling over the wall or bed or in the air, and will describe them. Other symptoms were mentioned by the speaker. Cases with which he had come in contact have simulated three conditions, locomotor ataxia, multiple neuritis and myelitis.

The speaker spoke of the beneficial results following the use of injections of salt solution. Dr. E. F. Wells had told him of a case which he believed to be practically cured by the use of large injections of salt solution.

Dr. Billings, in closing the discussion, said his belief is that a *Bothriocephalus latus* which will attack a red blood cell will not attack a tissue cell, but at present our knowledge of the blood conditions in these cases is in its infancy.

The clinical cases related by Dr. Church he had also seen, and in the original address which he delivered in Boston he added four other cases of the disease. He had not been able to procure a necropsy in the cases in which death occurred, but he gave their clinical histories. Dr. Church and he were associated in at least one of these cases, and he observed it through most of its clinical course—the one he referred to as terminating like an ascending myelitis. In addition, the patient presented in her first two years of illness a secondary anemia as marked as any he ever saw.

He had tried all sorts of treatment, and from the first had used the one suggested by Dr. Church, and while in some instances it had been of benefit, it was his experience that the patient dies, in spite of the normal salt solution in any amount injected into the cord. The only good influence it has is that it fills up the blood vessels. He did not think the portal circulation is improved very much, or the intestinal tract cleansed very much by washing out four or five feet of the colon and leaving the rest of the small intestine untouched.

# Periscope.

## DEUTSCHE ZEITSCHRIFT FUER NERVENHEILKUNDE

(Vol. 22, 1902, Parts 1-2.)

1. A Case of Stab Wound of the Spinal Cord (Brown-Séquard Paralysis) with a Special Consideration of the Ability to Localize. SCHITTENHELM.
2. A case of Brown-Séquard Unilateral Paralysis after Stab Wound of the Spinal Cord. FÜRNROHR.
3. Experimental Contribution to the Knowledge of the Inhibition of the Reflexes after Unilateral Section of the Spinal Cord. KRON.
4. The Histology and Pathology of Brain Tumors. BIELSCHOWSKY.
5. Remarks upon the Pathological Anatomy of Syphilis of the Central Nervous System. ERB.
6. Transplantation of Tendons in the Spinal Paralysis of Children. VULPIUS.
7. Clinical Contribution to the Knowledge of Hereditary and Family Spastic Paralysis. KÜHN.
8. Relations Between Myoclonia Familiaris and Congenital Myoclonia. LUNDBORG.
9. The Babinski Toe Reflex in Physiological Conditions. BICKEL.
10. Brief Communications: (1) The Polyclinic for Nervous Diseases in Strassburg. FÜRSTNER. (2) Lesion of the Conus Medullaris and Cauda Equina. ROSENFELD.

1. *Stab Wound of Spinal Cord.*—Schittenhelm reports the case of a man who was stabbed in the back between the fifth and sixth cervical vertebræ, with a broad-bladed knife. Immediately after the injury, however, the patient had complete paralysis of the right arm and leg, and difficulty in micturition and defecation. Several months later there was slight motor paralysis on the right side, and some sensory disturbances on the left, particularly loss of pain and temperature sense. A most careful description of the sensory phenomena is given and Schittenhelm discusses the exact localization of the injury. An interesting symptom was the condition of the localization sense. This was found to be completely lost in regions where motility, deeper sensation, and touch were intact, and when analgesia and thermalgesia were absent. It was variously disturbed in other regions. He believes the trouble was due to injury of the posterior columns and the cerebellar and motor lateral columns of the right half of the spinal cord.

2. *Brown-Séquard Unilateral Paralysis.*—Fürnrohr also reports a case of stab wound of the spinal cord causing Brown-Séquard's paralysis. Immediately after the injury the right leg was paralyzed, and there was a peculiar feeling in the right side as far as the nipple. Nine weeks later slight movement could be performed with the right leg. There was thermanesthesia in the left leg, the tendon reflexes were increased on the right side and slightly increased on the left side, and there was also ankle clonus on the right side. The tibialis and toe phenomena of Strümpell were both present in the right leg. A careful examination of the patient showed diminution of the pressure sense and

position sense on the same side as the motor paralysis—that is the right—and on the other side paralysis of the pain and temperature senses. There was also some peculiar subjective paresthesia on the right side.

3. *Hemisection of the Cord and Inhibition.*—Kron has performed a number of experiments upon dogs, making a hemisection of the spinal cord in the upper cervical region, then testing various reflexes in these animals in order to determine what effect this lesion had. The essential conclusions are as follows. After section of the spinal cord there is inhibition of the reflexes which is much more transient than is generally supposed. Therefore there does not seem to be any reason to suspect the existence of inhibitory fibers. After hemisection of the spinal cord respiration ceases on the corresponding side, but this is restored as soon as the phrenic nerve is cut upon the opposite side. The reflexes recur at varying intervals that depend largely upon the level and upon the severity of the lesion. There is no essential difference between the phenomena in animals and human beings; the differences are only slight.

4. *Brain Tumors.*—Bielschowsky reports four cases of brain tumor. The first occurred in a girl of eighteen years, who had nausea and headache, the latter becoming so severe that she was brought to the hospital, where bilateral choked disc was discovered. Later she had weakness in the legs, then staggering gait, loss of vision and some impairment in the heart. There was then loss of power in the right arm. She was re-admitted to the hospital and divergent strabismus was noted. There was also a distinct Romberg symptom and gradually increasing somnolence, then a series of convulsions, the patient finally dying. A tumor was found involving the corpora quadrigemina and apparently proceeding from the choroid plexus. There is a careful description of the histological changes including the appearance of the tumor, which was apparently epithelial in structure. The case was characteristic because paralysis of the muscles of the eye and ataxia were both present. The second case was a woman of forty-two years who had had symptoms of cerebral trouble for three years. There was headache, tinnitus, and attacks which consisted of tremor with numbness of both arms. There was impairment of vision and finally complete loss of sight. When admitted to the hospital she was found to be entirely deaf in the right ear; hearing was normal in the left ear, and taste and smell were not disturbed. There was papillitis in both eyes, weakness in both arms, and stumbling gait. She developed slight paralysis in the lower half of the right side of the face, which finally became complete, with some reactions of degeneration in the muscles. The patient finally died as a result of a bedsore. At the autopsy a tumor was found at the base of the cerebellum pressing upon the right side of the pons and destroying the right acoustic and facial nerves. This tumor appeared to be a fibroma and to arise from the pia. It is interesting that the hemiplegia was not alternating, it was homolateral. In a third case, a man twenty-seven years of age, the patient had noticed loss of power for half a year. There was some headache, vomiting, bilateral choked disc, staggering gait, and three days after admission, in the course of a severe attack of headache with frequent vomiting, the patient died. A glioma that had undergone cystic degeneration was found occupying the floor of the fourth ventricle. At the autopsy this was supposed to be cysticercus. The fourth case, a woman of twenty-four years, had suffered from vertigo for a number of months. later the gait was uncertain and staggering, and for about four weeks before admission she had had frequent vomiting. When admitted bilateral choked disc was present, there was tenderness over the left occipital lobe, and staggering gait, but no disturbance of sensation. The patient died suddenly, and at the autopsy a tumor was found in the right frontal lobe, cystic in character, which proved to be an

angioma. The interesting symptom in this case was the long persisting vertigo.

5. *Pathology of Nervous Syphilis.*—Erb, after discussing the manifold lesions and types of disease produced by syphilis, and reaching the conclusion in common with other pathologists, that it is impossible to state positively in many instances whether or not a lesion is syphilitic, has undertaken to collect a number of presumably syphilitic cases with degenerative lesions from the literature, and to classify them according to their different types. The first type includes cases of gummas, meningitis, myelitis, arteritis, with direct or focal degeneration of known specific character, in all of which leutic infection was definitely known or probable. The second class includes typical or primary direct degenerations not apparently specific associated with more or less certain specific changes in the meninges, the cord and the blood vessels. This includes a number of non-tabetic scleroses. Finally a third group consists of primary scleroses in certain syphilitic individuals without associated specific lesions. Of these he collects cases representing simple syphilitic disease of the pyramidal tracts, combined systematic disease, systematic disease of the posterior columns, especially tabes, and primary nuclear degenerations, atrophy of the eye nerves, etc. He summarizes his results as follows. That there are in some cases of unquestionable syphilis of the central nervous system changes that resemble primary degenerations, in other cases in which the primary degenerations form the predominant feature of the disease, there are slight syphilitic changes, and finally primary degenerations without syphilitic changes in patients who have certainly been infected with syphilis. He therefore believes that there is no reason for supposing that the absence of syphilitic lesions, as in the third group, is sufficient proof that the process is not syphilitic. In fact there can be no doubt that syphilis is frequently the cause of various forms of degeneration, particularly that involving the tracts. Regarding the nature of syphilis he suggests, as has often been suggested before, that the analogy between syphilitic and tuberculous lesions leads to the supposition that the infecting organisms must be very similar. It is interesting, however, to note how differently the two processes act upon the central nervous system, for there is no evidence that tuberculosis has ever been associated etiologically with either progressive paralysis or tabes. This seems to prove that the localization of the process in these diseases is not due to some weakening factors that act particularly upon the involved tissues.

6. *Tendon Transplantation.*—Vulpius discusses the transplantation of tendons in the spinal paralyses of children. This is of course useful only in partial paralysis, and may possibly be of use in other forms of paralysis such as that following injury as well as anterior poliomyelitis. He mentions some cases in which this transplantation was performed upon the thigh. In one instance the tendon of the semimembranosus was transplanted into the tendon of the quadriceps, at the same time the semitendinosus was cut in order to relieve the flexor spasm, and finally an osteotomy was performed in order to correct genu valgum. Seven months after the operation the patient was greatly improved and the gait was almost normal. Another case, a boy of six years and a half, had had poliomyelitis in the first year of his life, causing extensive paralysis in both legs followed by extreme deformities. A series of operations was performed to correct these deformities. The tendon of the extensor hallucis was transplanted to the tendon of the tibialis anticus; the tendon of the biceps was transplanted to the tibia, and finally after numerous tenotomies the leg was straightened. In order to promote extension from the knee, the tendons of all possible muscles were transplanted to that of the quadriceps. Seven months later the patient was able to walk without apparatus, and although there was

complete ankylosis of the left knee, there was a fair degree of flexion and extension in the right knee. There was also considerable increase in the power of the muscles and an extraordinary improvement in the condition of the patient.

7. *Hereditary and Family Spastic Paralysis.*—Kühn reports the following interesting instance of hereditary and family spastic spinal paralysis. Nothing could be learned concerning the grandparents of the patient, but the father had always—according to report—had some difficulty in walking. The mother had always been healthy. Of seven children, one, a daughter, died in infancy, two other daughters were married and had healthy children, a third emigrated, and the three sons suffered from a disease apparentlv similar to that described in the father's case. At the age of eighteen or nineteen one son began to experience pains in the spinal column in the region of the hips. Gradually the gait grew worse, but his arms remained healthy throughout, and he suffered from no other symptoms. He was married twice but had no children. Investigation showed rather typical spastic paraplegia, the electrical reactions of the muscles were normal, the patellar reflexes were increased, the Achilles tendon reflex and ankle clonus were not present, Babinski's phenomenon was not present, and Strümpell's tibialis phenomenon was pronounced on both sides. The second son also commenced to notice his disease about the eighteenth year. The symptoms were similar to those of his brother. The third son noticed the symptoms earlier, in the tenth or twelfth year. Kühn also reports a fourth case with somewhat similar symptoms. The patient began to notice symptoms of his trouble about his twenty-second year. His gait gradually became impaired and finally it was necessary for him to use a cane. The arms were normal, the muscles showed resistance to passive movements, the reflexes were increased, there was ankle clonus and Babinski's phenomenon was present. The tibialis phenomenon of Strümpell was present in both legs. This patient had four brothers all of whom died in infancy, and three sisters. His father had always been healthy. Kühn, in discussing these cases, states that it is difficult to understand what part heredity plays in the development of the disease. The anatomical lesion is probaby primary systematic degeneration of the lateral pyramidal tracts. The prognosis is not unfavorable to life, but it seems impossible to produce any effect unon the disease by treatment.

8. *Family and Congenital Myoclonia.*—Lundborg contributes a discussion of the relation of familial myoclonia to congenital myoclonia, giving the following interesting comparative table of the symptoms:

| CONGENITAL MYOCLONIA. | DEMENTIA MYOCLONIA OR FAMILIAL MYOCLONIA. |
|---|---|
| (1) A chronic disease probably caused by some autointoxication. | (1) A chronic disease probably caused by some autointoxication. |
| (2) Symptoms are present at birth or appear in earliest childhood, rarely later. | (2) Symptoms occur in later childhood, but not often afterward. |
| (3) Usually affects several persons of the same sex and often several brothers or sisters of same family. | (3) Affects several brothers and sisters of same family and not rarely several families in same group. |
| (4) Myotonic reactions. | (4) Sometimes myotonic reactions. |
| (5) Slight psychic disturbances may occur from time to time in various forms, as weak-mindedness. | (5) Pronounced psychic disturbances occur in course of the disease and terminate with distinct dementia. |

| | |
|---|---|
| (6) The striated muscles are affected by a tonic spasm which differs in intensity in different muscles and rarely affects those of the eye. | (6) The striated muscles are affected by a spasm which at first is tonic, later clonic. It varies in intensity in different muscles and the eye muscles are rarely affected. |
| (7) Alcohol has a favorable action in moderate doses. Fever also produces improvement, but cold and fatigue make the symptoms worse. | (7) The same. |
| (8) Mechanical irritation causes spasms of the muscles which gradually relax. | (8) The same. |

Lundborg believes that there are so many points of similarity in the two diseases, that the apparent intimate relation cannot be ascribed merely to accident. There is much evidence to prove that myoclonia is due to a poison generated somewhere in the system.

9. *Babinski Toe Phenomenon.*—Bickel has tested the Babinski reflex in more than 300 persons, mostly women and children, and found it positive whenever there was reason to believe in the existence of a lesion in the pyramidal tracts. It is also occasionally positive in hysteria, rarely positive in healthy adults, sometimes slightly indicated on one side. Occasionally it was positive at one investigation and negative at another. Often, however, in persons with a healthy nervous system in whom the reflex was negative while awake it became positive during deep sleep. Under these circumstances it was usually energetic and not slow as in pathological conditions. Also in cases of chloroform narcosis the reflex was found to be positive, and therefore Bickel concludes that it is an indication of functional inactivity of the cortex.

10. *Briefer Communications.*—Rosenfeld reports the case of a man twenty-eight years of age who received a severe injury in the lumbar portion of the spinal column causing immediate paralysis of the legs. Some months later on examination there was found to be increase in the reflexes in the lower extremities, the gait was impaired, there was loss of control over the sphincters, disturbance of sensation on the outer and upper sides of both feet, over the pubic and sacral regions and extending along the back of both thighs, that is, a typical saddle anesthesia. A diagnosis was made of a lesion of the conus medullaris.

JOSEPH SAILER (Philadelphia).

## ARCHIVES DE NEUROLOGIE.

(No. 85, January, 1903.)

1. The State of the Fundus of the Eye in General Paretics and its Initial and Final Anatomical Lesions. KERAVAL and RAVIART.
2. The Evolution of Obsessions and their Passage into Delusions. SÉGLAS.
3. Motor Re-education in Diseases of the Nervous System; its Application to the Ataxia of Tabetics. CONSTENSOUX.

1. *The State of the Fundus of the Eye in General Paretics.*—The author states that it is only in the second half of the nineteenth century, that, thanks to the ophthalmoscope, neuropathologists were able to examine the fundus of the eye of general paretics. Since 1853 Lasègue, Moreau, Marcé, Dagonnet practiced this examination, but left no data concerning the alterations they observed; Bouchut, relating three cases of general paresis, notes in each the existence of papillary atrophy. The author re-

ports the notes of Voisin and Magnan in 1868, of Tebaldi in 1870 and of Noyes in 1872. In 1879 the thesis of Boy and the memoirs of Doutrevente appeared, who, having examined 47 general paretics, came to the following conclusions: "Papillary atrophy either commencing or confirmed is met with from five to ten times in one hundred cases. We remark more often some vascular alterations, such as especially the dilatation of the veins, the alteration of the walls and some hemorrhagic spots or a generalized congestion of the fundus of the eye." The author gives the results of his studies of 51 cases of general paresis at the asylum of Armentiéres. He also gives full history of three cases and finishes his article of thirty pages (which contains 9 illustrations of sections made), with the following conclusions: "From the ophthalmoscopic examination of out of 51 general paretics, there appears the following: (1) the majority of these patients at the asylum of Armentières present some notable lesions of the fundus of the eye; there are scarcely any of them, except those who are in remission who do not present it; (2) we find in 7 patients, in whom general paresis has reached an advanced stage, 5 cases of white papillary atrophy, one of gray atrophy, one of bilateral posterior sclerotitis without myopia; (3) we find in 13 paretics a state of pale papilla, light in color; (4) in 22 others a mildly light appearance was observed in a segment of the papilla, whether external, internal, inferior or superior with undefined border. The two appearances are the preliminary states of papillary atrophy; (5) in general paretics in remission or attacked by a retarded form, it has been impossible to find any ophthalmoscopic lesion; (6) the ophthalmoscopic examination has in all points been confirmed by the microscope, it operates the same in the less accentuated cases, in papillitis and in neuritis presenting the same characters as in chronic diffuse meningoencephalitis: infiltration of the higher elements of the papilla and of the optic nerve by the connective tissue and neuroglia cells, thickening of the connective tissue of the nerve, diffuse alterations in the ganglion cells and the nerve fibers of the retina susceptible of ending in cases the most advanced, the total destruction of the nerve cells of the retina and the production of papillary sclerosis." There is subjoined a list of 29 authors who treat of the subject.

2. *Evolution of Obsessions and their Passage into Delusions.*—The author considers that the opinion generally entertained in France, that obsession does not evolve, does not transform itself, never ends in delusion properly so-called, is open to discussion. The author discusses obsession under the heads of melancholia, mental confusion, dream-like delusion and systematized delusions. One of the cases he observed is the following: Miss J. presents, since the age of eighteen, obsessions of doubt in the form of interrogations; metaphysical inquiries; later obsessional ideas in form of contrast, mania of blasphemy, impulses to blaspheme when praying in church, to strike the persons she loves best; doubt upon the execution of her divers impulses. At the age of twenty-four Miss J., who hitherto had full knowledge of her obsessions, comes to consider them as the action of an evil spirit. She, who hitherto, in analyzing herself, said very justly: "It is as if there was in me a second thought, which always contradicts me and urges me to evil, when I have the idea of good," finishes by believing herself really the sport of a wicked being, of a demon, which possesses her, opposes itself to all her acts, inspires and directs her at its will, and her conviction is such that she sees no succor except in exorcism. "According to Pitrés and Régis the forms which finally end in systematized delusion are obsessional states with symptoms chiefly intellectual, and those which turn into anxious depression are obsessional states with symptoms chiefly emotional, that is to say, phobias." "My personal observation," says the author,

"does not authorize me to partake of this view, applicable perhaps to some particular cases. I would rather believe that obsessions, which may finally end in characterized insane accidents, whatever may be the form, are chiefly obsessions called intellectual and, in particular, the different types described under the general name of *folie du doute.*"

3. *Motor Re-education in Diseases of the Nervous System; its application to the Ataxia of Tabetics.*—The subject is discussed under two heads: (1) Motor re-education in general, and (2) the re-education of the ataxic. It may be understood, says the author, that in cases where some sort of perturbation of the nervous system has deranged that delicate mechanism, without at the same time destroying completely the elements of the neuromuscular motor system and notably where voluntary contractions are not abolished, a new education of the altered centers can re-establish the equilibrium of the contractions and consequently the lost functions. Experience proves that this hope is legitimate. This is the first principle of motor re-education. It is a second which extends our field of action. No movement is effected without the simultaneous concourse of several muscles and at times, when one of them becomes insufficient, others may compensate its deficient action. He cites the cases of aphasic and agraphic patients, who may acquire special education. Re-education, the method based upon these two principles, can be defined as the totality of the processes of therapy, by methodical movements, which look especially to the re-establishment of functions, either by correcting, by a new impulse, anomalies of function in nervous elements or in attributing to other organs, functions belonging primarily to destroyed organs. At times function alone is disturbed, but in many other cases the organs themselves are injured and should themselves be treated by independent processes of re-education. It is necessary to associate other methods of physical therapy. In the matter of re-education general knowledge is only of value for the applications which one can make of it to each particular case. There cannot exist an immutable series of exercises applicable to all patients stricken with the same affection. It is necessary in order to direct the treatment usefully, after the diagnosis is established, to determine exactly the nature of the troubles observed, their localizations, their degree in each muscular group; thus only is one able to foresee what it is possible to expect of these muscles and what exercises of re-education suit them; then we should watch closely the effects, modify them according to the changes which are observed and seize from day to day the various indications which present themselves. We should take great account of the symptoms observed consecutively with the motor troubles, the disorders of sensation for example, the state of the forces, the intelligence and good will of the patient. To re-educate a patient is a work essentially clinical and consequently varies in each particular case, the success of which depends no less upon the ability of the physician than upon the symptoms which he has to combat. This is why we think that re-education can only be well undertaken by a physician and not by his assistants, and that by a physician having experience in nervous affections and knowing the difficulties of the clinic; by a specialist familiar with therapy by methodical exercises. Re-education may be applied to the following affections: (1) Static and locomotor; (2) movement of superior members; (3) troubles of phonation; (4) movements of the face and neck; (5) movements of the eyes; (6) respiratory movements: (*a*) functional troubles, tics and stammering; (*b*) troubles of organic origin, cardiac, emphysematous, tuberculous; (7) movements of the heart. The author gives the history of the re-education of the ataxic. The Swiss physician Frenkel obtained the first important results. He states that Frenkel's exercises were more empirical than scientific, but he first formulated the idea that ataxia regularly treated is not incurable. If we

have been hitherto powerless to re-establish reflex muscular action when abolished, we can still in a large measure diminish the number and frequency of involuntary movements. We know that muscular sensibility can supply articular sensibility, which is always more profoundly affected; that, save in grave cases, muscular anæsthesia with its divers varieties is rarely absolute; that muscular sensation can be ameliorated by re-education, thus become a great aid in the re-establishment of equilibrium. It is not rare to see in tabetics groups of muscles that in appearance are paralyzed and in which methodical exercises, little by little, permit patients to recover a more or less correct voluntary contraction. The first part of the treatment of the ataxic is the re-education of each muscular unit; the second part is the treatment of incoordination. What is most interesting, is the understanding of contraindications, for before undertaking a treatment long and difficult, we should be assured that it is not to be injurious and that it would really be useful to the patient. The contraindications are, *e. g.* cachexia of the subject, the existence of cardiac troubles, or asthenia very marked, the co-existence of certain symptoms, arthropathy, fragility of bones, amaurosis, psychical troubles, or even deficiency of intelligence. Since we understand better the treatment of tabes, we do not consider it an affection having an evolution fatally progressive. When we are aware of a period of improvement, after one of decline, then comes the moment to regain the whole or part of the lost ground and to repair the disorders which have arisen. The cases where re-education gives no results are explained in our opinion, by one of the following reasons; certain contraindications have been misunderstood, the tabes is aggravated, the patient is refractory or inattentive, or the re-educator is not equal to his task. The author insists on special experience in the physician, in exercises adapted to each case, in care that they be not too prolonged, but at the same time frequent; "little and often" is the rule. During the daily exercises, which the physician directs in person, he should give the patient the most minute directions as to the movements which he is to execute in his absence. It is well to interest an intelligent man in his treatment by giving him some explanations. The improvement of some of the patients excites his emulation and the sight of the troubles of others inspires some resignation. The author gives brief histories of 11 cases: The first, an officer in the army, forty years of age, confirmed tabes for seven years; motor incoördination for two years, had to abandon active service, walked with incertitude, assisted by a cane, with difficulty ascended and descended stairs, understood perfectly his treatment. At the end of four months there was scarce a trace of incoördination, he returned to his regiment, mounts his horse. Since the summer of 1901 he has not left active service. No. 2. At the end of four months rejoined his regiment. No. 3. At the end of three and a half months left the hospital alone and afoot. No. 4. Had been for seven months in bed and absolutely incapable of standing upright. At the end of the year she is able to take care of herself, provided she is prudent, but never dares walk in the street unaccompanied. "If only a few years ago tabes was in the great majority of cases considered a malady without appeal, almost fatally progressive and necessarily ending in confirmed ataxia, today, neurologists agree that such a picture no longer corresponds very often with the facts; not only does the malady spontaneously present durable remissions, not only must we settle with tabes frustes, when it only betrays some mild symptoms and does not evolve, but we know that we are not unarmed before the danger of posterior sclerosis; we know that our therapeutic methods result more and more frequently in arrest of the malady. Several physicians have also been able to speak of the curing of tabes, thus bringing together two words which hitherto seemed incompatible; it is to say, how changed is the prognosis of the malady; it is the same in the most grave of its symptoms, ataxia. Numerous observations prove that ataxia is not incurable; we can now oppose to it rational and efficacious therapeutic methods." RICHARDS (Amityville, L. I.).

### AMERICAN JOURNAL OF INSANITY

(Vol. 59, No. 2.)

1. Nature and Pathology of Myoclonus Epilepsy. CLARKE and PROUT.
2. Nurses in Hospitals for the Insane. RICHARDSON.
3. The Mental Status of Czolgosz, the Assassin of President McKinley. CHANNING.
4. Litigious Insanity, with Report of a Case. LANE.
5. Sanitation in Asylums for the Insane with Especial Reference to Tuberculosis. MAC CALLUM.
6. Some Results and Possibilities in Family Care of the Insane in Massachusetts. COPP.
7. Tent Life for the Demented and Uncleanly. WRIGHT.
8. Tent Life for the Tuberculous Insane. HAVELAND.
9. Sympathetic Insanity in Twin Sisters. WALKER.

1. *Nature and Pathology of Myoclonus Epilepsy.*—It is an essentially rare affection, only 57 cases have as yet been recorded, which include the authors' four. Many conditions have been described, which are unquestionably related to this disease type, if not such in reality. Unverricht was the first to definitely describe it in 1891. Probably many cases have escaped observation. Myoclonus has been found to be associated with general paresis and other cortical lesions. The predisposing causes of myoclonus are found largely in family degeneracy, essential epilepsy equally so in this respect and hence occurs in several generations of the same family, and it is doubtful if myoclonus-epilepsy ever occurs in an individual of healthy stock. Various forms of degeneracy may lead up to myoclonus-epilepsy and other manifestations of degeneration, like insanity, chorea, etc., which are always found in other members of the family. The stigmata of degeneration are well marked in such families. The immediate exciting cause alleged may be trivial. Evidence of traumatism to the head or other parts of the body or fright is found in a small percentage of cases. Fatigue from overwork, alcoholic debauches and infectious fevers occasionally present. These are doubtless of serious moment, only in so far as they induce a condition favorable to autointoxication. This applies especially to extreme fright. The autointoxication may be external to the intestinal tract, is more probably dependent on a condition of faulty chemotaxis of the cortical cells. Men are more often affected, in the ratio of five to three. All the recorded cases developed prior to twenty, except one at thirty. In three-fourths of them between nine and fifteen. In about half of the cases the epilepsy appears first, in one-third the myoclonus, in the balance the two simultaneously. The epileptic attacks usually nocturnal at first, later diurnal. As a rule epilepsy continues through life, but in a few instances exists only at the beginning of the disease. The seizures are never preceded by an aura, but premonitory signs of increased myoclonic contractions are in evidence, yet seizures may not occur at their climax. The epileptic attacks are often abortive, the tonic stage being absent, tongue-biting and post-epileptic coma are also wanting. The muscles involved in the epileptic seizures are most often those affected by the myoclonic contractions. There is no periodicity in the occurrence of the seizures and death from status epilepticus unknown, yet a sort of myoclonus status often hastens or actually causes death. Long remissions in the epileptic paroxysms often occur, years even intervening. Many times consciousness is only disordered or partially lost and post-epileptic automatism rare. Mel-

ancholic mania quite common after a number of "bad days." Epileptic mental stigmata present. The myoclonus here is often atypical The contractions are lightning-like and of a fibrillary character, involving only parts of certain muscles. The "live flesh" or tremors develop more or less rapidly in a few weeks or months into typical myoclonic contractions. They rarely remain fibrillary throughout life. A single tonic contraction may constitute the whole clinical picture. The clonic contractions of myoclonus pass imperceptibly into the tonic stage of the epileptic paroxysm, the contractions are often strong and affect large masses of muscles, so that locomotor effect is common. The trunk is often affected, causing the body to be jerked antero-posteriorly and laterally. The face and distal portions of the extremities are quite often involved. Both sides of the body are affected alike, but the contractions may be more frequent and violent on one side. The myoclonus usually begins in one of the upper extremities, then involving the lower, chest, abdomen, neck and face in the order named. The muscles about the eyes and mouth usually last affected, although the reverse has happened. Tongue and diaphragm often suffer in severe cases, causing a peculiar grunt. The contractions more severe some days than others, and it is rare for days to pass entirely free from myoclonus. As the disease develops the myoclonus becomes more or less persistent while awake, causing an exhaustion often terminating fatally. Long remissions may occur under sedative treatment even in severe cases. At first the myoclonic contractions may be controlled voluntarily or by calling into play antagonistic muscles. In the early stages the convulsive movements only occur during voluntary movements. When the disease becomes fully developed all voluntary movements are diminished, a feeling of lassitude is often complained of. Both superficial and deep reflexes are usually exaggerated, especially on the side most affected. Speech disorders are not always present. As a rule there is a lack of physical and psychical development. Rachitis, a dwarfish stature and lack of muscular and osseous development. A general malnutrition results from the difficulty in swallowing; the patients are also anemic and subject to gastro-intestinal symptoms, anorexia and diarrhea readily occur and regurgitation of food is common to some cases. Aubuminuria is occasionally found, indicanuria is frequent. The psychical state varies from slight mental enfeeblement to complete imbecility or idiocy. The majority are feeble-minded, often emotional and subject to attacks of furious violence. Many become insane with unsystematized delusions. Dementia progresses with the disease. Prognosis invariably bad, no cures are reported, yet the disease may be ameliorated. It tends to shorten life, the patient generally dying of inanition, pulmonary congestion and a sort of presenility due to the malnutrition. The majority die in early adult life. These patients are usually sterile, but children born die early. The diagnosis of typical cases is easy. Errors are generally due to placing too much stress on single symptoms of the disease. There are family types and sporadic cases, the latter forming the majority. The disease is to be differentiated from hystero-epilepsy, essential epilepsy with pseudomyoclonic contractions or "jerks," the idiomuscular tremors in epilepsy, isolated or multiple tics in epilepsy, choreic epilepsy and myoclonia in general paresis. The state of consciousness often decisive with respect to essential epilepsy. The stigmata of hysteria should give rise to a doubt in the diagnosis. Loss of consciousness, tongue-biting, irresponsive, dilated pupils and stertor confirm the diagnosis of epilepsy. Short, sharp contractions of the proximal muscles of the extremities and especially those of the trunk, which cannot be produced voluntarily, indicate the myoclonic nature of the trouble. The pseudomyoclonic movements or premonitory "jerks" in essential epilepsy always attended by impairment or loss of consciousness, are simple flexure movements and never multiple, affect the upper extremities only, are always bilateral and synchronous and consist either in flexion of the forearm or adduction of the whole upper extremity. In the common atypical cases the differential diagnosis is difficult and probably forms the connection between classic and myoclonus-epilepsy. The idiomuscular tremors of epilepsy are rhythmical, uninfluenced by the will, and never severe enough to have locomotor effect. The various tics of epileptics are generally confined to the face, not coördinated and usually cause voluntary or reflex emotional acts. Coprolalia, echolalia, achemesis and fixed ideas are often associated. A close association of the pathogenesis of the two affections probable; the difference consists in the motor neurones involved. In choreic paresis and infantile spasmodic hemiplegia the convulsions begin and generally involve the parts once paralyzed, the choreiform movements are generally rhythmical and confined to one-half of the body; unilateral atrophy also occurs. Some authors believe essential myoclonus is so closely related to degenerative chorea that a differentiation is impossible, but this is hardly tenable. It is rare to see any form of chorea associated with epilepsy, while twenty per cent. of myoclonics are epileptic. The writers claim never to have seen a case of choreic epilepsy in their extensive experience with epilptics, and in all probability myoclonus-epilepsy and choreic movements are easily differentiated from myoclonus. The treatmen can only be palliative, of which bromides occupy the first place and should be given for a long time. Flechsig's opium treatment has met with good results. Chloral may be combined with advantage. As myoclonic contractions are most severe in the morning, a light repast may be given the patient before arising with advantage. Coffee in large portions has warded off threatened myoclonic contractions. Attention should be given to the diet and general hygiene. An outdoor life is advisable as far as practical. The lesions underlying myoclonus, as well as those of epilepsy, must be in the brain cortex in all probability. The gross anatomical appearances are of little account. The pathology of the few cases observed shows a mild meningitis and an accumulation of cell elements about the vessels and cells, with some increase of the interstitial product common to degenerative processes of the brain and meninges. The lesion seems to involve the nucleus and intranuclear network of cells of both sensory and motor types. The pathogenesis seems to be an intoxication or autointoxication of these cortical cells probably due to a faulty chemotaxis of these cells. Three cases are reported to confirm the data given above.

2. *Nurses in Hospitals for the Insane.*—Nothing new is offered; the general experience of hospital officers is confirmed. The advantage of female nurses on the male wards is shown by the greater neatness, less frequency of complaints and the patients' greater contentment.

3. *The Mental Status of Czolgosz, the Assassin of President McKinley.*—A full detailed account is given of the assassin as related by members of his family, friends, associates and those with whom he had lived at various periods of his life. The writer holds that he was suffering from a form of mental disorder similar to the hebephrenic type of dementia præcox and was wholly dominated by the delusion of benefiting "the poor people," which led to the tragedy. He summarizes his argument in the ten following conclusions: (1) I feel that from further information than that possessed by those experts who examined Czolgosz after his crime, the opinion then expressed by them cannot be accepted as the final one. (2) Owing to lack of time, it was impossible in the examination referred to, to investigate the early history of Czolgosz. Had this been done some of his statements would have been found to be inaccurate. (3) He was not in my opinion an anarchist in the true sense of the word, and while anarchist doctrines may have inflamed his mind and been a factor in the crime, it was not the true cause or an adequate explanation. (4) He had been in ill health for some

years, changing from an industrious and apparently fairly normal young man into a sickly, unhealthy and abnormal one. (5) While in this physical and mental condition of sickness and abnormality, it is probable that he conceived the idea of performing some great act for the benefit of the common and working people. (6) This finally developed into a true delusion, that it was his duty to kill the President, because he was an enemy of the people, and resulted in the assassination. (7) His conduct after the crime was not inconsistent with insanity. (8) His history for some years before the deed, the way in which it was committed and his actions afterwards furnish a good illustration of the typical regicide or magnicide as described by Regis. (9) The postmortem examination threw no light on his mental condition and would not invalidate the opinion that the existing delusion was the result of disturbed brain action. (10) Finally, from a study of all the facts that have come to my attention, insanity appears to me the most reasonable and logical explanation of the crime.

4. *Litigious Insanity, with Report of a Case.*—This condition has been classed with paranoia by some authors, by others as a form of moral imbecility; the latter is untenable, as moral imbecility is manifested at an early age, while the former usually appears at about the third decennary, further it shows a progressive character, a fixed idea generally dominates the whole life and is woven into the delusions. There may be a real or fancied grievance as the starting point and this so controls the patient's thought, that he begins a legal action to obtain redress, and as his ideas of his rights are perverted, the courts fail to sustain his position and after repeated failures to get justice he fancies due him, he arrives at the conclusion that the legal profession, the officers of the court, including the judges, are in a combination to defraud him. He then resorts to any means to gain his end and he becomes so absorbed in his ideas, that he neglects his business, family and his personal wants and comforts. His final appeal is to the chief executive. The writer reports the case of a lady whose litigious delusions so dominated her, that she accused her husband and children of charges she always failed to sustain, involved herself in various suits at law, ruining herself financially, and finally lived in the most abnormal manner, and at last was found dead in her home. Various efforts had been made to have a guardian appointed for her, but always unsuccessfully.

5. *Sanitation in Asylums for the Insane with Special Reference to Tuberculosis.*—The obligation of isolating the tuberculous insane in institutions is urged, as well as providing them with commodious, sunny and well ventilated quarters. Verandas should be provided for bed-ridden patients and all the precautions of personal hygiene exercised to prevent dissemination of the infectious material.

6. *Some Results and Possibilities in Family Care of the Insane in Massachusetts.*—A true estimate of the value of family care in Massachusetts has been prevented by the unsettled state heretofore of the authorities governing and directing it, causing a lack of continuity of policy and sustained support essential to its best development and proper test. The law limits the price paid for board to $3.25 per week and requires that the State patients be visited once in three months at least, and these patients may be removed from their boarding place or returned to the State Hospital at the discretion of the board, if abused, neglected or found to be unsuited to this system. The system has had various vicissitudes, at one time the number increasing and then declining until recently, when it has remained stationary for a number of years. The largest number placed in any one year was 83 and the lowest 4, with a yearly average of 34.9. The average number during the whole existence of this system is 119.43, the largest number at any one time was 179, the smallest 94. Some of the patients, 53 or 45 per cent., have been out from ten to sixteen years; 29.1 per cent. have

been found unsuitable. Some remained less than one month, the average duration is one year ten and a half months; 10.9 per cent. were returned on account of illness, as only the quiet, tidy, tractable, healthy and helpful patients are available for this system, the hospital being obliged to retain the objectionable class of chronic patients. There has been no dearth of applications for boarders of this class and 83 per cent. of those inspected were approved, 313 families having received patients. The average duration of boarding in one family is three years one month. Very rarly have detrimental effects on the family been observed, but on the other hand beneficial, that is, the premises have shown greater neatness, house become better furnished, with other evidences of thrift, and in some instances an attachment to the patients has been observed. Eighty per cent. of the boarders are women. Nearly all forms of mental disease are represented in these patients, in the chronic stage of course. Few mishaps have occurred. A saving to the State of forty-seven cents a week per patient is shown, but this is not a fair comparison, as the patients would tend to lessen the hospital per capita by increasing the number under treatment and the work they might do, thus saving the employment of extra help. The advantages of the system have not as yet been demonstrated, still the author sees in it advantages that may be made manifest in the future.

7. *Tent Life for the Demented and Uncleanly.*—Twenty patients were selected of whom 60 per cent. were bed-ridden and most of them stupid and demented with no appreciation of their condition or surroundings. All of the patients showed a gain in weight within three weeks, except one who weighed the same; but he subsequently gained slightly, with a marked improvement in his habits, and when the camp was closed he had gained seven pounds in weight, had greatly improved mentally and was tidy in his habits. A number of other instances are cited where there was a marked improvement in the mental and physical conditions, as well as in the habits and general deportment and of the whole number only three continued filthy at the time of closing the camp. All manifested greater interest in their surroundings and occupied themselves in reading, games, etc. The average gain in weight was 13 3-5 pounds; the highest 50 pounds, the lowest 6 pounds. On the whole the experiment was a success and justifies its further trial.

8. *Tent Life for the Tuberculous Insane.*—The patients treated consisted of 40 suffering with tuberculosis in the Manhattan State Hospital, East. Forty square feet of floor space was allowed to each patient, ventilation was amply provided, as well as bathing facilities. Asepsis was carefully looked after and all means used to destroy the bacilli. The medical treatment was purely symptomatic, the chief reliance being placed on hygienic measures and diet in particular, four meals being served daily of plain, wholesome food with a liberal allowance of milk of good quality. On the approach of cold weather, heating by means of stoves was provided and there was no difficulty in maintaining an even temperature, even in the severest weather. The results of the treatment are shown by reducing the percentage of deaths from 17 4-5 to 8 4-5 of the total number in the hospital, being the lowest in the history of the institution. Eighty-one cases in all were treated in this way; of these 55 showed an average gain in weight of 6½ pounds while 24 showed an average loss of 4 2-3 pounds and 2 no change in this respect; 8.64 per cent. of the cases treated recovered and were those in the incipiency of the disease, clearly showing the importance of its early diagnosis. The most apparent benefit was manifested in the increased appetite and ability to assimilate the food. Night sweats were conspicuous by their absence. Besides the benefit to the patients affected with the disease on admission, this treatment tends to prevent infection of other patients, who are in a more or less susceptible condition

owing to their physical condition incident to the attending mental disease. Further the mental condition of these patients was improved, although the majority were in a state of chronicity. The results attained have led to providing for an extension of the system.

9. *Sympathetic Insanity in Twin Sisters.*—In the sisters' prior life they had shown the tendency to be affected with the same ills almost simultaneously and their bond of sympathy was very strong under ordinary conditions. No history of heredity is noted, but on one becoming violently maniacal while away from home, her sister was sent for, who then at once became similarly affected and hospital treatment was necessary for both, where they were separated and not permitted to see each other; still their disease seemed to run a very similar course, an improvement occurring in each simultaneously, and a relapse occurred in each at the same time and each has become demented, in which condition they are at present.

McCorn (Amityville, L. I.).

### BEITRAEGE ZUR PSYCHIATRISCHEN KLINIK

(Vol. 1, No. 3. November, 1902.)

1. The Measurement of the Motor Accompaniments of Psychical States. R. Sommer.

This is the only original article in this number of the Beiträge. The rest of the number is occupied by two book reviews. The author in his introduction to this study says that since the idea of psychophysical parallelism has become something more than a simple hypothesis, movements of expression, the effects of psychical states, must be studied by every means available. These motor reactions he takes up for study in three groups: First, muscular; second, vasomotor, under which he includes secretory, and third, electromotor; and his paper is accordingly divided into three parts as follows:

1. *Measurement of the Physiognomic Movements of Expression in the Musculature of the Forehead.*—For the purposes of this study the forehead was selected as presenting the results of the activity of only two muscles (M. corrugator supercilii and M. frontalis). The contour of the folds in the skin of the forehead was studied by applying smoked paper. This leaves the soot marks where the creases occur and rubs it off in the intervals between them. The results of this method showed that these creases were the effects of muscular contraction and were most marked on the side which was morphologically the more strongly developed.

The rest of this section is devoted to the description of an apparatus for studying the forehead muscles in motion. It consists essentially of a disc applied to the forehead, the movements of which are transferred by levers and Marey drums to a revolving smoked disc where are two pens, one of which records the vertical, the other the horizontal movements.

2. *Measurement of the Vasomotor Changes in the Skin.*—For these measurements the usual means employed has been the plethysmograph. The author, however, makes the general criticism of the use of this instrument that the changes in the volume of the arm are an end result and not necessarily attributable altogether to vasomotor changes but in part probably to muscular contractions. He therefore considers it desirable to study the vasomotor changes as they present themselves in the skin alone. To this end the author has devised an apparatus. It consists of a capsule which is fastened on the forearm by removing the air from it, thus requiring no straps. Into the top of this capsule is fitted another with flexible walls having connected with it a tube containing illuminating gas which goes to a burner. Any change of volume in this second capsule, due to change in the quantity of blood in the skin, alters the size of the gas flame. The light

from this flame is thrown by a parabolic mirror on a selenium cell which is attached to a galvanometer. The deflections of the needle of the galvanometer therefore show the changes in the volume of blood in the skin. These changes may be recorded in a smoked drum by having the electric current connected through a solenoid with a recording needle.

3. *The Measurement of the Electromotor Changes in the Fingers.*—This part, like the former two, is taken up mostly with the description of an apparatus consisting in the main of two electrodes on which the hands or fingers are rested and which are in circuit with a mirror galvanometer. The author concludes that the electromotor processes are stronger in the fingers than in the palm of the hand. These results are largely the effects of muscular contraction and in so far as they represent involuntary movements of expression they can be said to be an electromotor end result of a psychophysical process. WM. A. WHITE (Binghamton, N. Y.).

### VOPROSSY NERVON-PSYCHICHESSKOY MEDITZINY

(January—March, 1902.)

1. Rudolph Virchow. I. SIKORSKY.
2. A New Clinical Variety Idiophrenia Paranoides. I. SIKORSKY.
3. From the Domain of Psychopathic Literature. P. PREOBRAJENSKY.
4. The Pathological Anatomy of the Nerve Cells as Applied to Mental Diseases. I. IVANOFF.
5. Paralytic Rabies in Man as Modified by Antirabic Inoculations. I. HMELIEVSKY.
6. Suicide in Keeyeff. N. OBLONSKY.
7. Traits of Character and Mimicry in Dementia of Alcoholic Origin. I. SIKORSKY.

1. *Biographical Sketch.* 2 *and* 3, *Psychopathic Literature.*—Two very interesting contributions to the comparatively new study of the subject of psychopathic literature as exemplified by the type of writers whom Lombroso designates as mattoids—graphomaniacs. As is well known these are not all confined in lunatic asylums, and some of them not only occupy a more or less prominent place in certain European literatures, but are proclaimed as heralds of new tendencies hitherto unknown in the world's literature. Sikorsky coins for this class the new term of Idiophrenia (Greek, *idios*, one's own, peculiar, *phrenes*, mind), while paranoides points to the presence in these literary decadents of ideas of grandeur in connection with those of persecution so characteristic of paranoia, which these cases simulate very closely. The subjoined examples of some of the degenerate scribbling show clearly how perfectly sane and eminently intelligent critics may be betrayed into taking very seriously, senseless inanities of diseased minds that are dished out before the gullible public as the brilliant scintillations of the "moderns," the "symbolists," and similar rubbish.

4. *Pathological Anatomy of the Nerve Cells.*—A history of the inception and development of the various methods employed in the study of the nervous system.

5. *Modified Rabies.*—As based upon investigations of his own cases and those found in the literature of the subject, the author draws the following conclusions: (1) There is at times observed in persons bitten by mad dogs and treated afterward by Pasteur inoculations, a peculiar affection of the nervous system with the predominance of symptoms of meningomyelitis in the thoracic or lumbar portions of the cord. (2) This affection bears the character of an infectious disease, as it is usually accompanied by a rise of temperature, at times by respiratory disturbances, and frequently by paralysis of the facial and oculomotor nerves; its duration is from one and a half weeks to two months, always terminating favorably. (3)

Its clinical history, its course and the analysis of the etiological data do not permit of its being classified with any known variety of meningomyelitis. (4) The picture of the disease corresponds exactly with that of the paralytic variety of rabies in man. (5) In view of the above and also because of the common etiological factor we may assume with a great degree of certainty that we have here to deal with a paralytic, being a form of rabies which becomes considerably weakened in its intensity by the aid of antirabic inoculation.

6. *Suicide in Keeyeff.*—A continued article.

7. *Traits of Character and Mimicry in Dementia.*—A series of portraits of alcoholic individuals, showing the acquisition of certain characteristic traits undoubtedly due to the abuse of spirits with all its baneful consequences. Considering the prevalence of drunkenness—secret as well as open —this fact is one of immense importance. Many a writer's literary work carries with it the reflection of his spiritual self as influenced by the excessive indulgence in alcohol, and a close study of some of the pearls of the modern literature (special reference is made to the decadent school of writers) reveals a peculiar style which the author justly calls "alcoholic."

(April—June.)

9. Psychiatry and Psychology. Zieben.
10. White Dermography. P. Nikolsky.
11. Affection of the Motor Neurone in Tabes. M. Lapinsky.
12. A Review of the Institutions for the Care of Insane in Russia. A. Kotzovsky.

9. *Psychiatry and Psychology.*—An historical survey of scientific psychiatry and its intimate connection with experimental psychology.

10. *White Dermography.*—It has long been observed by several investigators (Rayer, Käbner and others), that in certain individuals a trifling irritation of the skins causes blushing, which may last for a considerable time, and Dr. Jardin-Beaumetz has demonstrated a case of a woman on whose skin a phrase could be written which would remain in letters raised above the surface for many hours. In some cases the red color would gradually give way to a white line, a condition somewhat rarer than the former one. The author ascribes this phenomenon to the fact that in white dermographism the vasodilator nerves of the skin are insensible and do not respond readily to irritation, while the vasoconstrictor fibers are strongly and quickly irritated, thus causing contraction of the vessels and pallor of the irritated region. These results of irritation as applied to the skin are observed not only when the irritation is caused mechanically, but also, as verified by the author, when brought about by electricity and heat. Barthelémy found red demographism in epilepsy, hysteria, tabes, gout, exophthalmic goiter, disseminated sclerosis, general paralysis, and also in various skin affections as psoriasis, scleroderma, pityriasis, urticaria and others. As regards white dermography the author thinks it a rule in prurigo, and also in chronic eczema, erythema scarlatiniformum, pemphigus ichthyosis.

11. *The Motor Neurone in Tabes.*—A continued article.

12. *Insane Asylums in Russia.*—A continued article.

(July—September.)

15. The Motor Neurone in Tabes.
16. The Insane Asylums in Russia.
17. Multiple Births. A. Inossoff.
18. Suicide in Keeyeff. A. Obolensky.

15. *The Motor Neurone in Tabes.*—Some of the results of observations

and experimental investigations by the author are embraced in the following conclusions: There are cases of tabes in which the motor and trophic affections of the muscles are due not to a neuritis, but to a primary alteration of the cells of the anterior horns of the spinal cord. The following scheme presents the clinical and the pathological differences between the two varieties of tabes:

| AMYOTROPHIC TABES WITH AFFECTION OF THE ANTERIOR HORNS. | AMYOTROPHIC TABES OF NEURITIC ORIGIN. |
|---|---|
| The paralysis does not correspond with the distribution of the nerve roots. The paralyses and the atrophies are limited to regions connected with certain muscles supplied by certain nerves; within the affected area there may be healthy muscles. | The various paralyses and atrophies are localized in correspondence with the branches of individual nerve roots; both paralysis and paresis are to be found in the area supplied by the affected nerve. |
| The atrophies and paralyses are asymmetrical. | There is a certain symmetry observed in the affected muscles of the various extremities. |
| The affected muscles showed fibrillary contraction. | No fibrillation in the attacked muscles. |
| The pareses and paralyses are preceded by muscular atrophy; it is only with the appearance of the latter that the patient begins to feel weakness. | Paralyses appear before atrophies. |
| Electrically there is a quantitative diminution of irritability. | There is observed a reaction of degeneration in the affected nerves and muscles. |
| Microscopically we find atrophic changes of individual fibers in the affected nerves and pronounced alterations in the anterior horns. | There are degenerative changes in the nerve roots, but the anterior horns remain normal. |
| The motor neurone affection is rather of an atrophic than inflammatory character and its course is subacute. | |

The affection of the anterior horns is either diffuse or focal; in this latter we find no typical signs of poliomyelitic anterior; there is no inflammatory condition so characteristic of that disease. Both the focal and the diffuse affections may be met with in the same cord; we find at the same time contracted capillaries, which tend to cause anemia of the anterior horns, limited, however, because of the very small size of the capillaries, at first possibly to individual cells, or small groups of the same; so that the neighboring cells or collections of cells may remain quite normal and unaffected. The various paralyses, pareses and atrophies and in general the muscular affections of spinal origin originating in a certain segment of the trunk or extremities and accompanied by changes of pain or temperature sense in the corresponding portions of the body must be regarded as segmental affections of the spinal cord; on one hand there were attacked the anterior horns, on the other hand the sensory posterior fibers entering the central gray matter. These so-called "segmental affections" are demonstrable clinically in the simultaneous affection of the motor and sensory spheres of certain parts of the body.

16. *The Insane Asylums in Russia.*—An historical review and particu-

lar description of the most prominent insane asylums in Russia.

17. *Multiple Births.*—A rather curious attempt to classify multiplicity of births, anthropologically arranged as regards the population of Russia, as one of the symptoms of racial as well as individual degeneration.

18. *Suicide in Keeyeff.*—The greatest number of suicides occurred during the hot months of June and July; this fact goes far toward substantiating the proposition held by many neurologists that high temperature acts as an irritant to the nervous system which frequently results in attempted suicide; it is, however, the barometric pressure that seems to play the most important rôle. ALEX. ROVINSKY.

## MISCELLANY.

### REFLEX CONVULSIONS IN GROWING BOYS AND GIRLS. E. S. Smith (Lancet, Jan. 24, 1903.)

Convulsive attacks occur in rickety and highly neurotic infants during teething and other reflex irritations. It is not so well known that they also occur as pure reflexes during worry in children about eleven or twelve years old, members of families of distinct neurotic tendencies. There is one symptom in common deserving attention, namely, habitually cold feet in these individuals. It usually provokes little attention, but, if disregarded, may thwart the best efforts of the physician in treatment. With these cold feet, the child's sensibility to chills is increased. He can offer no effectual resistance to the sudden changes of temperature. Digestive derangement follows, malnutrition, and weakness and injurious tendencies are irritated. The nervous system is then readily thrown off its balance. If such nervous conditions may be controlled in the infant, they certainly may be in the older child. Such nervous seizures are as harmless in the older child as in the younger child, and apparently leave the patient in no worse condition, but there are cases where the condition becomes so chronic and recurrent that hardly any appreciable excitable cause may exist to bring them about. It is therefore advisable that the children should have their health built up, and such sickness and general depression as coldness of the feet should always have attention. JELLIFFE.

### THE PATELLAR TENDON REFLEX IN CROUPOUS PNEUMONIA IN CHILDREN. M. Pfaundler (Münchener med. Woch., 1902. Vol. 49, p. 1,211).

From a study of 200 cases of croupous pneumonia in children Pfaundler found that in 27.5 per cent. the patellar reflex was absent, or diminished. From this study he concludes: (1) As a rule fever was well marked, but the absence of the reflex was noted in certain cases which were at the time non-febrile, and the sign occurred not uncommonly after the crisis, the temperature being normal or subnormal. (2) It is seen only exceptionally in children over ten years, and rarely in infants at the breast. (3) A relationship between the part affected and the condition of the reflex cannot be made evident. (4) Many of the cases showed severe general symptoms, especially an initial cerebral affection, whilst the mortality (5.4 per cent.), was rather high for that age-period. (5) It was particularly met with in strongly built and well-nourished children. (6) The course of the pneumonia in these cases showed no unusual deviation from the normal. (7) Apparently the sign arises early in the disease, and sometimes it was observed when the physical examination of the lung was quite negative. (8) As a rule the reflex returns during or soon after the crisis, and generally, though not always, at the same time on the two sides.

In 87 cases diagnosed as lobular pneumonia Pfaundler found that, excluding one moribund and one doubtful case, the patellar reflex was absent only on two occasions, and diminished on three, and in all of these cases the consolidated patches were confluent. He is of opinion that the loss or diminution of the tendon reflex is due to the action of specific toxic substances on the nerves at the periphery and at the reflex center. The sign may be of service in the differential diagnosis of pneumonia from meningitis.

JELLIFFE.

THE CRIMINAL RESPONSIBILITY OF THE EPILEPTIC. John Punton (Med. Record, Nov. 15, 1902).

The author draws the following deductions: (1) That epilepsy is a symptom of some brain disease; (2) that its continual presence tends toward mental deterioration; (3) that mental responsibility of the epileptic depends upon the extent to which the mind or self control has been impaired by the epilepsy; (4) that the legal test of insanity is not sufficient, as mental irresponsibility is not incompatible with a knowledge of right from wrong; (5) that epileptics are to some degree at least, responsible for criminal acts, especially when the epilepsy is produced by their own fault; (6) that criminal acts of epileptics appeal to medicine rather than to law for their proper adjudication; (7) that in all cases of murder in which epilepsy is proven the law should be amended to allow of like commitment to an insane hospital rather than to the penitentiary; (8) that the mental responsibility of the epileptic should be referred to a medical commission appointed by the court, which again may be referred to local or county medical societies to name its members.

Universal medical experience indicates that well established epilepsy is a sure sign of biological inferiority. But it is absolutely necessary to demonstrate in each case the extent to which mind control has been impaired by the epilepsy, as well as the existence of epileptic insanity. All authorities agree that irritability, impulsiveness and aversion to control are the marked characteristics of the epileptic psychoses. In true epileptic mania, the criminal act is usually unpremeditated, motiveless and accompanied by impairment of consciousness and temporary loss of memory. Homicide is its legitimate product. W. B. NOYES.

ACUTE POLIOMYELITIS AND ENCEPHALITIS. F. E. BATTEN (The Lancet, Dec. 20, 1902).

Upon the question as to what is the cause of the changes noted in these two diseases in the nervous tissues concerned, and especially as to the part in that cause played by thrombosis, the following points are offered. It has been stated by those who support the view that the condition is primarily inflammatory, that many observers have occluded the vessels of the spinal cord for varying periods, and examined the changes so produced, but, although they found marked chromatolysis of the nerve cells, they did not find perivascular exudation or hemorrhage. Furthermore, they state that Dr. Leonard Hill ligated the cerebral arteries in monkeys and the brains of these animals were examined by Dr. F. W. Mott at various periods after the operations, who found extreme chromatolysis of the cortical cells, but no inflammatory change or hemorrhage. Such an argument sounds almost conclusive, and would at once negative the view that there is primary thrombosis, but it must be remembered that obliteration of a large vessel is by no means necessarily comparable to the condition which is produced by thrombosis occurring in smaller vessels. Far more to the point are the experiments of Prevost and Cotard with regard to the changes which take place in an infarcted area. By injecting fine tobacco powder into the vessels, they succeeded in getting infarcts in various organs, and they proved by these experiments that congestion, hemorrhage, and exudation of cells were the early result of obliteration of small vessels. Having therefore established the possibility of such a condition as is found to be present in these cases of acute encephalitis and poliomyelitis being due to a primary thrombosis, the next point to show would be that the pathological process may be limited to the distribution of a single vessel. If one therefore studies the vascular supply of the spinal cord it is found that the gray matter of the anterior horns receives its chief supply from branches of the anterior median artery, the white matter being chiefly supplied from vessels arising from the pia mater. The branches of this anterior median artery supply the whole of the gray matter of the anterior horns, but not that of the posterior horns. In the spinal cord from cases of infantile paralysis the line of demarcation between an affected and unaffected part is sharply defined and limited to the gray matter of the anterior horns—that is to say the area of congestion, or, in the later stages, of necrosis, corresponds very closely to the area of the blood-supply of the anterior median vessel. It cannot be asserted that the process is always thus accurately limited, for it is well known that the vessels in the ventral portion of the spinal cord and outside the gray matter are often engorged and possibly thrombosed, and again in the gray matter small areas of softening are found which do not occupy the whole of the gray matter. Such conditions are easily explicable, since congestion of vessels commonly occurs around an infarcted area and the localized softening in the gray matter is due to the thrombosis occurring in the smallest vessels. The microscopic appearances of the softening of the gray matter of the spinal cord correspond closely with that due to thrombosis of small vessels in the brain. The very frequent occurrence of anterior poliomyelitis in the lumbar sacral region seems to be another point greatly in favor of the view that the condition is primarily vascular, for it is well known that the gray matter of the anterior horns of the lumbar region is situated at a point most peripheral from its blood supply, *i.e.*, the blood derived from the vertebral artery has to travel the whole length of the anterior median artery, and, further, it has been shown by Moxon that the reinforcing arteries of the roots do not readily assist the supply of that part of the cord. These, then, are the points in favor of the disease being due directly to a primary vascular thrombosis; in favor of the view that the disease is of direct bacterial origin may be quoted its prevalence during a certain period of the year, becoming epidemic, as in the present year, and its occurrence in several members of a family in its various forms, either cerebral or spinal, of which a most interesting example has been published by Dr. W. Pasteur. None of these points, however, are antagonistic to the view upheld by the author, for this infection only supplies the cause of the blood change. Furthermore, in cases of spinal infection that can be shown to be due to bacterial invasion, the changes met with are not limited to the gray matter, but affect gray and white matter alike. The author's arguments have been drawn chiefly from the pathological examination of the spinal cord, as the opportunity for examination of that part of the nervous system most frequently occurs. But what has been said in regard to the nature of the lesion applies equally to those cases in which the medulla, pons, cerebellum, or cortex is affected, the lesion being a primary thrombosis of the finer and terminal arteries. In conclusion, though there are many gaps to be filled up by further pathological investigation, he trusts that he has given grounds for considering cases of acute encephalitis and acute poliomyelitis as clinically identical; pathologically they are identical. The more frequent recognition of these cases of encephalitis will explain many cases of so-called meningitis in which recovery more or less complete takes place. JELLIFFE.

A CASE OF EPILEPSY, APPARENTLY OF NASAL ORIGIN. Robert Craig (Montreal Med. Jour., April, 1902).

Patient, male, nineteen years old, had suffered from "nose bleed, headache and epileptic fits for five years." During the six months previous to treatment of naso-pharyngeal condition, which consisted of small ulceration in the right nostril on the anterior half of the septum, associated with a large septal spur pressing on the posterior half of the middle turbinal, and accompanying congestion in nose and pro-pharynx, the convulsive attacks became much more frequent and of increasing severity. Since removal of the obstruction, a period of one year, there has been no recurrence of headache, nose bleeds or epileptic attacks. Hack and others have reported similar cases exhibiting the relationship between nasal disease and epilepsy.

J. E. CLARK (New York).

THE SILENT FORMS OF EPILEPSY. William P. Spratling (N. Y. Med. Jour., Oct. 10, 1902).

The greatest medico-legal problem connected with epilepsy is encountered in its purely psychical forms, unaccompanied by any motor disturbance. No other disease of the nervous system calls for so accurate a knowledge of cerebral localization as epilepsy. The silent forms of epilepsy come from some disturbance in parts of the brain known as the "organs of the mind," presumably the frontal lobes. Two kinds of epilepsy may thus develop,—one the psychomotor epileptic equivalent, the other the psychical attack pure and simple. These mental seizures are apt to occur in persons of a neurasthenic type. People who forget in a striking and unusual way, who disappear for long periods of time and who find themselves with returning consciousness in a distant place, undoubtedly suffer from epilepsy of this nature. They have done nothing violent, there has simply been a lapse in conscious operations of the mind without any violence on the part of the body. Cases of silent epilepsy are not infrequent and are of great medico-legal importance. W. B. NOYES (New York).

HYSTERIA AND ORGANIC DISEASE. Charles L. Dana (Med. Rec., Sept. 20, 1902).

The main objective stigmata and subjective symptoms of hysteria major are (1) the anesthesias; (2) the contractures; (3) the palsies and tremors; (4) the attacks, and (5) the peculiar mental state. The hysterical syndrome is oftenest associated with organic lesions due to trauma, but various symptom groups appear also with tumor of the brain, encephalitis, meningitis, multiple sclerosis, tabes and more rarely with inflammatory disease of the nerves. The different symptom groups are of variable stability. The anesthesias are the most mobile, fugacious and amenable to remedial measures. The paralyses indicate a deeper and more stable affection. They show a greater tendency to remain fixed. Contractures may be fugacious and irregular, but when established are even more obstinate than the paralyses. They do not disappear during sleep or moderate narcosis. The following case is reported by the writer: A woman had jumped from a window while drunk, and on admission to the hospital was delirious and paraplegic. Autopsy subsequently demonstrated a fracture of the first lumbar vertebra, and of the lowermost segment of the sacrum. The injury to the cord was slight, involving only its posterior portion. The symptoms were flaccid paraplegia, absence of knee-jerks, and anesthesia to the third dorsal level on the left, and twelfth dorsal on the right. The right side of the face and tongue were anesthetic. There was total amblyopia of the right eye. A case of a man crushed in the Grand Central tunnel accident, sustaining fracture of the tenth rib, contusion of the right knee, and a severe burn. Two weeks after leaving the hospital he developed a tremor and general nervousness, and failure of memory. He is unable to walk much and has vertigo. He has rigidity of the right hand. Spots of anesthesia over face. Another case of hysteria and tabes combined, the functional symptoms being evident by hysterical crying, hyperesthesia of the chest, and other sensory symptoms not explainable by tabes alone.

W. B. Noyes (New York).

MYASTHENIA GRAVIS. F. G. Finley (Montreal Med. Jour., July, 1902).

The author reports the details of an interesting case of this comparatively rare disease which was treated at the Montreal General Hospital. The patient, a male, forty-nine years old, no evidence of syphilis, alcohol very moderate, has always enjoyed good health up to the present illness. Occupation, engine driver. Eight to ten years ago had injury to scalp and severe blow on head in a railway accident, full recovery from same. Family history negative. Present illness began September, 1901, with weakness in arms, then of legs, and later in muscles of jaw. Difficulty in swallowing, particularly solids. Thickness of speech, and dribbling of saliva from lips, which was early noted. Loss of weight, attributed to inability to take sufficient nourishment. Symptoms vary in intensity from time to time, and patient is usually better in the morning than evening. Memory and intelligence are good. Motor power in arms and legs not diminished. Muscles medium sized and flabby. Inability to whistle well. Tongue protrudes to the right, slight tremor of same. Soft palate hangs to right and is flabby. Slight weakness of adductors of vocal cords, more on right than left. Difficulty in swallowing, at times regurgitation. Pupils, fundus, ocular muscles negative. Knee-jerks slightly increased. Plantar reflex normal. Slight thickening of radial arteries, abdominal and thoracic viscera normal. Temperature for sixteen days, while at hospital, subnormal (95 1-5 deg. F., to 98 1-5 deg. F.). Pulse 64 to 84. Marked changes from day to day, as to weakness, noted in the bulbar muscles. At times almost entire absence of weakness, no difficulty in swallowing or thickness of speech. Muscles responded readily to faradism, no diminution after repeated stimulation. Diagnosis based on weakness of bulbar muscles and marked variations of power from day to day and improvement exhibited after night's rest, particularly of swallowing. Early involvement of jaw suggestive of pseudo type rather than true bulbar palsy, where it occurs in the more advanced stages. Slight degree of weakness, greater on right than left, in protruding tongue, lessened degree of adduction of right vocal cord, *etc.*

J. E. Clark (New York).

A CONTRIBUTION TO THE TREATMENT OF SPASMODIC WRY-NECK. George R. Elliot (N. Y. Med. Jour., Oct. 11, 1902).

The writer analyzes a form of wry-neck which is purely a nervous disease. It is characterized by a spasm of the muscles supplied by the spinal accessory and sometimes by the upper cervical nerves. The patient is usually neuropathic and of bad heredity. The sternomastoid and trapezius muscles are the ones usually involved. The head is inclined to the affected side by the trapezius, the chin is raised, and the head is rotated to the opposite side by the sternomastoid an dtrapezius. The spasm is intermittent. These cases have not always been cured by nerve resection and muscle cutting. The author advises an apparatus with a chin piece, a rotating spring, and two uprights. It is movable and restricts the spasm.

W. B. Noyes (New York).

SOME OBSERVATIONS UPON DELUSIONS, IMPULSIVE INSANITY AND MORAL IDIOCY. Bernard Oettinger (American Journal of Medical Sciences, December, 1902).

The question of sanity should be based more on the individual's demeanor and conduct in its entirety, or the collective reaction to slight stimuli rather than upon expression of any single delusion, however bizarre. Impulsive insanity should include those forms of degenerative insanity wherein the subject is possessed of morbid propensities and impulses and wherein, if the latter be consummated by action, the same is undertaken without a clear motive, but because an irresistible impulse so to act is experienced. Moral insanity is a form of imbecility characterized by the absence or enfeeblement of those emotions which combat the inconsiderate gratification of egotism. What Pinel termed *manie sine delire* may be resolved into three classes: (1) Certain forms of degenerative insane states, impulsive insanity and moral idiocy, both as a rule unassociated with false beliefs; or where, like the aura of epilepsy, the hallucination has no causal relation to the reflex movement; (2) instances in which the subject is possessed by delusions, but these were slight and associated with reasoning power; (3) cases of active melancholia, in which the constant dread of something unknown but fearful is translated into a violent act. The idea of moral insanity was extended by Pritchard to include a moral perversion of the natural feelings, affections, inclinations and impulses, without any remarkable disorder or defect of the intellect and particularly without any insane illusion or hallucination. The writer believes that the phrases moral and impulsive insanity should be avoided. The best authorities today look upon the mental types which they represent as degenerative forms of mental alienation. W. B. NOYES.

URBAN SELECTION AND MENTAL HEALTH. J. A. Macpherson (Review of Neurology and Psychiatry, Feb., 1903, No. 2).

The author presents a short statistical note bearing on the physical changes of type consequent on urban selection. The first is an increased dolichocephaly, the second point bears on the variation in height, in some cities the country inhabitants are taller, in others the urban population. An increased "brunetteness" is thought to be a further result. The selected urban type then is tall, dolichocephalic, dark haired and dark eyed. Mentally it is vigorous, quick-witted, capable of endurance, and self-controlled. Fond of amusement, it is not readily overcome by excitement, and does not yield itself easily to excesses. As regards alcohol it ought to have undergone a special evolution. The immigrant fresh from the country is confronted by all the sordid features and must run the gauntlet of zymotic disease, alcohol, syphilis and tuberculosis. On the question of insanity it is not clear whether the author thinks that there is more insanity in urban populations than in the country. The tendency of the argument seems to be that the many factors in city life tend to raise the incidence of mental deterioration. JELLIFFE.

THE PROMINENT EYE. A. Haig (Med. Record., Oct. 11, 1902).

The underlying cause common to Grave's disease and minor conditions of prominent eye is high blood pressure, and this the author holds is due to the presence of uric acid in the blood. Only rarely do new growths or myopia cause prominence or retraction of the eye. The prominent eye is often met with in cases of high blood pressure (migraine), headache, enlargement of the thyroid occurs in most women during menstruation when the blood pressure is raised; the quick pulse is met with in all high blood pressure conditions. With the prominent eye is associated a puffiness or distended condition of the skin, which may later relax. Conditions which hinder the circulation through the lungs, such as bronchitis, asthma, and emphysema, increase the prominent eye; also coughing, straining, heavy exertion, or mental excitement. The prominent eye tells us whether there is high blood pressure, how long it has been there, and whether the heart has slowly and steadily hypertrophied or not.

W. B. NOYES (New York).

**THE DIFFERENTIAL DIAGNOSIS OF MULTIPLE SCLEROSIS.** B. Onuf (Onufrowicz) (Brooklyn Med. Jour., Nov., 1902).

In speaking of the diversity of opinion as to the pathology of this disease, the author states that it may be due, in part, to various cases presenting differences in the anatomical process, or dissimilar stages of the disease having been observed. He suggests that a better understanding may be obtained by the careful analysis of a great number of autopsy cases with founded with multiple sclerosis. The pathological processes of the diffuse sclerosis, pseudo-sclerosis, syphilis, hysteria, general paresis, multiple softening foci, tabes, combined system disease, transverse myelitis and Brown-Séquard paralysis are most likely, according to his experience, to be conand pseudo type are much the same as in the insular form, but the latter is differentiated, anatomically, by localization, not being chiefly confined to the hemispheres. Thus, clinically, a preponderance of mental symptoms would be somewhat indicative of the first mentioned disease. Cerebral syphilis, in its multiplicity of symptoms, may resemble multiple sclerosis. A correct diagnosis, oftentimes, necessitating a careful general, as well as special examination. Although of value, too great stress should not be given to the subsidence of symptoms under anti-syphilitic treatment, unless rapid (two weeks) and with marked improvement. Multiple sclerosis, without treatment, oftentimes exhibits a similar, but less speedy tendency. Typical scanning speech, is strongly diagnostic, while nystagmus is not pathognomonic. If well developed, without ocular defects present, it points decidedly in favor of disseminated sclerosis. Because of their constancy, the author regards facial expression and mimicry, also emotional liability, as evinced by the great tendency either to laughter or crying, to be two symptoms of cardinal importance in differential diagnosis between insular sclerosis and syphilis; not general nor specific pseudo-paresis. Optic neuritis, often, only symptom in incipient stage of disease. May almost clear up later on. Syphilis usually attended with more profound retinal and choroidal changes than multiple sclerosis. Evidence of past iritis favors diagnosis of syphilis in doubtful cases. Seventh and eighth cranial nerves not uncommonly found affected in disseminated sclerosis. Multiple cerebral softening, at times, strongly resembles disease. Senility and vascular changes differentiate in favor of cerebral softening foci. The speech disturbance, emotional liability and dementia of bulbar palsy may simulate multiple sclerosis. Symmetrical bulbar atrophies, more gradual onset, and symptoms pointing to an involvement of the anterior horns and lateral tracts aid in forming a correct diagnosis.

J. E. CLARK (New York).

**ANGIO-NEUROTIC EDEMA.** W. E. Deeks (The Montreal Med. Jour., July, 1902).

Patient, female, eighty-three years, asthmatic, with chronic cardiac disease. Urticaria at times. Prior to angio-neurotic attack, health quite good, although taking suprarenal extract because of threatened cardiac edema which appeared upon cessation of remedy. On morning of attack, urticarial swelling under one eye disappeared quite quickly. At noon, while in usual state of health, patient became dyspneic ; constant irritable coughing and swelling of face. Whole face, including ears, cyanotic and swollen. Eyes decidedly prominent and bulging. Tongue edematous, speech almost inarticulate from preceding cause. Marked swelling of parotid and submaxillary glands. Pulse not affected to any great extent. General pulmonary asthmatic condition. No febrile disturbance. Large veins of neck not engorged. Thrombosis of deep jugular veins associated with cardiac affection considered probable, but rejected because of immediate relief afforded by hypodermic injection of morphine and atropine. Cyanosis disappeared, and in few hours patient comfortable. In twenty-four hours the swelling of tongue and lips had disappeared. Case interesting because of locality; association with asthma, possibly with urticaria; and promptness of relief by morphine and atropine, *etc.* Suprarenal extract administration not supposed to have had any connection with causation of attack, because of immunity in its use, prior to, and since attack.

J. E. CLARK (New York).

INEQUALITY OF THE PUPILS IN THORACIC ANEURISM. R. C. B. Wall and E. W. A. Walker (Lancet, July 12, 1902).

After discussing the matter at some length and citing a number of cases, with evidence based upon clinical observation and experiment, the authors sum up their conclusions as follows: The explanation ascribing the anisocoria which sometimes occurs in cases of thoracic aneurism to interference with the sympathetic, is unsatisfactory. On anatomical grounds: (a) because in the majority of cases there is no evidence of implication of that portion of the sympathetic nerve trunk containing pupil dilator fibers, and (b) because it is not established that sympathetic filaments supply the sac wall of an aneurism. Further, alterations in vascular conditions may be associated with alterations in the size of the pupils, low arterial tension being associated with large pupils, and vice versa, phenomena for which the spiral structure of the vessels of the iris is probably responsible, and, therefore, local inequalities of blood-pressure may be associated with inequalities of the pupils. Clinical evidence has shown that enlargement of pupils is often associated with diminution of the temporal and radial pulses on the same side of the body, and obstruction of the carotid artery on one side of the neck is associated with enlargement of the pupil on the same side. By experiment upon the carotid artery in rabbits it was found that its obstruction caused enlargement of the pupils on both sides, owing to the freedom of circulation at the base of the brain, and that injection of water into the carotid artery of a dead rabbit caused enlargement of the pupil on that side. In the human subject digital compression of the carotid was also associated with enlargement of the pupil on that side. Their final conclusion was that inequality of pupils associated with thoracic aneurism is usually due to inequality of blood pressure in the ophthalmic arteries resulting from the abnormal vascular conditions. JELLIFFE.

TREPHINING FOR BRAIN TUMORS. Joseph Ransohoff (Jour. of American Med. Asso., Oct. 11, 1902).

Operable brain tumors are rare. Only six per cent. of all cases are suitable for operation. The writer reports two successful cases. The first had suffered from convulsions beginning in the foot, rapidly extending over the side, followed by unconsciousness. He had violent headaches and frequent attacks of vertigo. Fundus shows some papillitis. Operation showed a tumor over the right foot center the size of a hen's egg. Recovery, with occasional light epileptic seizures, and a slight paresis of the arm and leg.

A second case of removal of a solitary tubercle from the ascending frontal convolution on account of muscular twitching and occasional loss of consciousness. No headache at any time. The patient recovered, with only a moderate amount of weakness of flexors of thumb and fingers. To make a diagnosis of cerebral tumor without headache is unusual. Against 104 cases of brain tumor in which operations were successful, there are 157 in which the operation was unsuccessful. Subcortical tumors particularly ofter escape attention. If the dura and cortex are not involved headache often fails. W. B. NOYES.

**Pathology and Treatment of Epilepsy.** William H. Thompson (N. Y. Med. Jour., Nov. 8, 1902).

Epilepsy cannot be defined as a convulsive disease, for convulsions are at most only occasional effects, not inherent adjuncts to epilepsy. Nor can it be defined as a cerebral disorder producing loss of consciousness, for even that is not invariable. Aphasia alone may be the only symptom. The fact of epilepsy does not depend at all on the number or on the variety of the symptoms, but solely on the question whether the symptoms are epileptic or not. Epilepsy is a specific disease *sui generis* protean in its manifestations. The first and simplest symptom is part of the epileptic process as much as the latest and most violent symptom. The never varying element in epilepsy is suddenness. Epilepsy is the only sudden disease. The first law of the physiology of the nervous system is that the beginning of every nervous action is always on the afferent side. A spontaneous, *i.e.*, a primary motor efferent discharge is unknown in physiology. Why should it occur in epilepsy? It is by the constant repetition of the same recurring excitation that certain groups of neurones become disciplined to react uniformly to a certain definite afferent excitation. This implies the establishment of a definite inhibition. Inhibition is solely the resistance begotten by habit to any irregular efferent response. Epilepsy is a disease characterized by a sudden derangement of the normal regulative inhibition existing between cortical nerve centers, induced in the first instance by an abnormal excitation. This shifts the primary seat of epilepsy from the motor efferent to the sensory or afferent portions of the nervous structure. As regards treatment therefore all epilepsies should be regarded as caused by some source of abnormal afferent excitation. Such a cause may be intracranial focus of irritation following injury to the head, venous thrombosis in the meninges, following typhoid or scarlet fever, or sunstroke, and a long continued dosing of one-twenty-fifth of a grain of mercury biniodide, t.i.d., and the application to the nape of the neck of bromide ointment is advisable.

Irritation of the nose, tracts of respiration or deglutition are frequent causes of epilepsy, and can be treated. Over excitability of the throat leading to bolting of food may be treated by application to the whole pharynx of nitrate of silver, ten grains to the ounce, followed later by tincture of iodine. To prevent bromism in cases needing continued bromide, give cod liver oil and phosphorus, or reinforce and reduce the bromid by coal tar drugs, such as antipyrin. Auto-intoxication and toxemia in some form, whether alcoholic, uremic, are the commonest causes. Open air treatment is as much indicated in epilepsy as in tuberculosis. The writer claims a cure in 70 per cent. of the cases of epilepsy.

W. B. Noyes.

**Alcoholic Epilepsy.** T. D. Crothers (Journal American Medical Association, Dec. 13, 1902).

The writer emphasizes the following facts: (1.) Alcoholic epilepsy is increasing rapidly in this country. (2.) It is a psychosis and neurosis which should be recognized, having distinct symptoms, the recognition of which is imperative in the treatment. (3.) When the toxic symptoms are convulsive and explosive and come on suddenly, the future of the case is very ominous and the warnings from these symptoms should be heeded. (4.) The connection between mixed drinks and these spasmodic symptoms is traceable and should be considered in the prognosis and treatment.

The cases are classified as follows: (1) The convulsive and maniacal types; (2) the demented and confused form; (3) the trance automatic and psychic classes. In the first, intoxication of a sudden maniacal type is followed by muscular explosions of extraordinary character. These attacks are followed by profuse stupor and often convulsive twitchings of the voluntary muscles.

In the states of dementia and confusion epileptic symptoms are prominent. The writer believes that the convulsive trembling and unconsciousness point to epilepsy which may be the cause instead of the effect. In the class of automatic trance and psychic cases certain blanks of memory occur, followed by automatic acts which the person never remembers. Narcotism by alcoholism or epileptoid symptoms may be found. Besides these there may be sudden alterations of personality and conduct and automatic trances. Thus a moderate drinker may perform some act of which he is unconscious and in an epileptoid state which may be regarded unusual or insane. W. B. NOYES.

EPILEPSY: ITS TREATMENT. Daniel R. Brower (Journal of the American Medical Association, Jan. 17, 1903).

Hygiene of the epileptic. This should be disciplinary, pedagogic, and dietetic. All stimulating narcotics should be eschewed. Differential care for the dullards and the over-bright should be carried out. The diet should tend to minimize nitrogenous food, permitting meat once a day, excluding all indigestible foods, and cutting down fat and sugar.

Drugs. If bromides are used, vary their administration by changing the vehicle. For children the dose is a grain for each year of their life, not exceeding 20 grains three times a day of bromide of sodium for an adult. If this fails add fluid extract of *Solanum Carolinensis*, beginning with a halfteaspoonful, increasing to two. In cases of cardiac enfeeblement the fluid extract of *Adonis vernalis*, in dose of from a half to five minims may be added. Coal tar drugs may be used, or belladonna. For the anemic cases bromide of iron may be used.

If irritability of the bladder ensues,, bromohydric acid or bromipin may be used. If the bromide treatment fails borax in ten grain doses. Oxide of zinc in doses of three to five grains, or strychnine may be of service. Santonin in increasing doses may be used. Combinations of salicylate of soda and antipyrin may be used if bromides disagree. Nitroglycerine in certain cases is helpful. W. B. NOYES.

PATHOLOGY OF TABES. L. Nageotte. (Presse Médicale, Dec. 10, 1902, No. 49).

Tabes, the author says, is an inflammatory disease which attacks a certain group of sensory and motor nerve roots as they emerge from the subarachnoid space. The process is allied to a syphilitic meningitis. The lesion is not solely a syphilitic one, but there is an interstitial transverse neuritis of the nerve root that has traveled up from the meninges and involved the interfascicular connective tissue. The posterior nerve roots are first involved and then the posterior columns throughout. The shorter fibers seemed to suffer more than the long ones. There is a progressive atrophic change in each fiber with thinning of the myelin sheath. Later the myelin disappears, the axis cylinder persisting.

JELLIFFE.

MASSAGE FOR TABETICS. M. G. Constentoux (La Presse Médicale, Dec. 6, 1902).

The author arrives at the following conclusions: (1) The employment of massage for tabes should be especially adapted for each particular case. (2) Massage though given with greatest care, and helpful in certain cases may be harmful to other cases. (3) It should be applied either by a physician acquainted with tabes, or by a masseur absolutely obedient to instructions. (4) Massage for tabes is indicated as follows; (a) for a general tonic; (b) as a means to combat certain sensory disturbances; (c) against certain complications. (5) Massage of the skin is useful in many of the sensory troubles, and also as a general tonic of extensive utility against nutritive changes. (6) The massage of muscles is without effect on the ataxia; it causes fatigue if too vigorous. It accomplishes little when there is paralysis or true atrophies. In slowly healing fractures in tabes it is valuable. (7) Passive motion is contraindicated when the joints are already too movable and when there is hypotonia of the muscles. (8) The active motions should be of a nature to re-educate the limbs. (9) Whatever be the conditions, the manipulations of massage should be gentle, and the seances brief when applied in tabes. W. B. Noyes.

Autopsy in a Case of Adiposis Dolorosa. F. K. Dercum and D. J. McCarthy (American Journal of the Medical Sciences, December, 1902).

The protocol of the autopsy was as follows: Superficial abdominal fat three and three-quarter inches thick. Loose in the fat were several hemolymph glands. The pia and brain were normal, except an abnormal arrangement of the third frontal convolution. The pituitary body was closely adherent to the *sella turcica,* which was converted in part into a tumor mass, which was an adeno-carcinoma. The thyroid gland was normal. The kidneys were in a condition of acute parenchymatous nephritis. There was an interstitial neuritis of the nerve filaments of the subcutaneous fat. The testicles were undeveloped. The symptoms that had been caused by that were obesity, large masses of fat accumulating on the abdomen and chest, which were extremely painful. As the deposit of fat grew the patient became excessively weak and easily fatigued. Four or five years before he had had an epileptic seizure, which recurred at intervals of a month or two. It was accompanied by an aura, biting of the tongue and unconsciousness. The knee-jerks were normal. There were no areas of anesthesia or hyperesthesia. The eyes showed very slight ptosis of the left side and some weakness of the internal rectus. Death was caused by erysipelas. W. B. Noyes.

Treatment of Epilepsy. L. Cerf (L'Anjou Médicale, 9, 1902, July).

The author reports in four cases of epilepsy treated by thyroid with such excellent results and he suggests that in all cases of so-called essential or idiopathic epilepsy that some form of thyroid therapy be instituted in the event of there being some thyroid dystrophy in the case.

Jelliffe.

# Book Reviews

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HANDBUCH DER PATHOLOGISCHEN ANATOMIE DES NERVENSYSTEM. Herausgegeben von DR. E. FLATAU, in Warschau; Privat Docent DR. L. JACOBSOHN, in Berlin, and Privat Docent DR. L. MINOR, in Moskau. First Abtheilung, Bogen 1-20. S. Karger, Berlin.

This is the first portion of a work on the pathological anatomy of the central and peripheral nervous system that is to be completed in 3-4 parts. As this portion is 320 pages, the completed book promises to be an important and extensive treatise.

In no branch of medical science has research been so active and profound as in that of the study of the nervous system, and the numerous observations made by pathological workers in many laboratories make it desirable that a collection of this scattered and discrete knowledge be made within the bounds of one work. This task the editors set for themselves, for in the preliminary announcement the names of Anton, Ballet, v. Bechterew, Benda, Brasch, Bruns, Cassirer, Cramer, Darkschewitsch, Elschnig, Friedimann, v. Gehuchten, Goldscheider, Heller, Hoche, Homen, Joachemsthal, Jolly, Luce, Lugaro, Mendel, Michaelis, Nonne, Oppenheim, Petren, Pick, Raymond, Rossolimo, Schlesinger, Stroebe and Weber, appear as collaborators. Surely an extensive and representative group of authorities.

The opening chapter deals with the modes of investigation of the nervous system, giving at great length the details of technic. This is one of the best presentations of this subject yet given. It is contributed by Jacobsohn of Berlin. Particular stress is laid on the subject of artefacts and their characteristics.

Dr. L. Michaelis gives a thorough chapter on the bacteriological investigation of the nervous system.

A very fine chapter is that of von Gehuchten's on the Pathological Anatomy of the Nerve Cell. It is well illustrated, splendidly written and an authoritative and modern exposition of our knowledge on this subject.

The general Pathological Anatomy of the Nerve Fibers is taken by Lugaro. In this chapter the important work of Kennedy, Ballance and Stewart is not considered;—certainly a serious omission. Lugaro also writes on the pathology of the neuroglia.

M. Nonne and H. Luce write on the pathological anatomy of the vessels. This is a masterly chapter, marred only by the author's almost slavish adherence to German literature.

Chapter VII on the Diseases of the Bony and Membranous Brain Coverings, by H. Stroebe, is just begun in this fascicle. From the publishers' standpoint, the work is most excellent, and if the succeeding parts are as comprehensive and able as this one the work will certainly meet with the success that its merits demand. JELLIFFE.

GESCHLECHT UND KRANKHEIT—GESCHLECHT UND ENTARTUNG. By Dr. P. J. MÖBIUS. Carl Marhold, Halle.

These are two contributions by this well known author to the subject of the differences in the character of the sexes.

The first brochure discusses in an interesting manner those diseases that are peculiar to men or women and also those affections that are apt to be more common in one sex than in the other. He makes two divisions. Diseases with natural sex differences and diseases associated with social sex differences. Under the former are included as diseases in males, hemophilia, muscle weakness, diabetes, leucemia, hay fever, brain tumors and congenital heart lesions; as female, chorea, whooping cough, torticollis, goiter, myxedema, Basedow's disease, scleroderma, chronic articular rheumatism, hysteria, migraine, manic depressive insanity, alcoholic neuritis.

In the second group he distinguishes as female belongings in the way of disease, cholelithias, acute yellow atrophy, wandering kidney, gastroptosis and trachoma. Man seems to suffer on account of his social position as all of the rest of the diseases that flesh is heir to passed to his account. The deductions are both interesting and suggestive.

In the second brochure a number of facts bearing on various anomalous degenerations are discussed. Cases of hypospadias, gynomastia, hermaphroditism, etc., are described and their philosophical relations pointed out. Both brochures are of interest and of not a little practical import.

R. Brown.

Der Achillessehnenreflex und seine klinische Bedeutung. Von Dr. A. v. Sarbo. S. Karger. Berlin.

This is a small brochure of 43 pages, devoted to the study of the Achilles jerks in health and disease. The first 11 pages are occupied with the details of the modes of investigation, and in the following pages the character of the Achilles jerk in hemiparesis, syphilis, mercury poisoning, articular atrophy, lead poisoning, alcoholism, ischias, tabes, paresis, and poliomyelitis are recorded.

The brochure is well considered, and is an excellent and convenient summary of knowledge of this reflex.

Jelliffe.

The American Year Book of Medicine and Surgery. Medicine. W. B. Saunders & Company, 1903.

The year book for 1903 is much better in every way than in previous years. Not that the room for improvement was so noticeable, but that the general excellence of the work has been enhanced by a greater attention to the details of the abstracts furnished and a few new chapters have been added that have greatly enriched the book. This is notably true in the chapter devoted to Physiological Chemistry edited by W. Jones and Reid Hunt.

For the neurologist the main interest will center about the chapters on Nervous and Mental Diseases, and Legal Medicine. The former contributed by Dr. A. Church, the latter by John Marshall and J. H. Rhein. Dr. Church has collected most of the truly good things done throughout the year, particularly in the domain of neurology. The abstracts on Insanity are not representative.

The work is improved also in its general attractiveness. The paper, print and binding are all that could be desired. Altogether it is the best year book published in the English language, and is a worthy addition to the library.

Walsh.

# News and Notes

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**Dr. Paul Flechsig** has been appointed an honorary member of the Jurjen University of Dorpat.

**Because** of the illness of Dr. Anton Bunn, of Munich, S. Hans Gudden will deliver the lectures in psychiatry.

**Prof. Remak** has been appointed Professor Extraordinary.

**Privat Docent E. Schultze** (Andernach) has the title Professor.

**Dr. L. Pierce Clark** has been appointed Consulting Neurologist to the Manhattan State Hospital at Central Islip.

**Cocaine in Georgia.**—At a recent trial in Atlanta, Ga., it was brought out that more than 3,000 prescriptions for cocaine had been filled in two months.

**Dr. E. Hitzig**, of Halle, because of a progressive affection of the eye, will be relieved from duty until September, after which time it is announced he will resign as Director of the psychiatric and nerve clinic. Dr. Hitzig founded at Halle the first psychiatric clinic in 1885. He was born in Berlin in 1838, in 1875 went to Zurich and in 1879 went to Halle.

**Congress of French Alienists and Neurologists.**—The thirteenth Congress of this society will assemble at Brussels on August 1, 1903, under the presidency of M. v. d. Bruggen. The program will include the discussion of Catatonia and Stupor, introduced by Mr. le Dr. Claus of Antwerp; Histology of General Paresis, by Mr. le Dr. Klippel; Treatment of Agitation and of Insomnia in Mental and Nervous Diseases, by Dr. Trenel. Dr. J. Crocq is the Secretary, to whom all communications should be addressed, 27 Rue Palmerston, Brussels.

**American Neurological Association.**—The Council announces that the Twenty-ninth Annual Meeting will be held in Washington, D. C., in conjunction with the Congress of American Physicians and Surgeons, on May 12, 13 and 14, 1903. There will be one session daily from 9 a. m. to 1 p. m. The sessions will be held at the Arlington Hotel. The Council desires to call attention to that part of Article VII of the Constitution which reads as follows: "The reader of a paper shall not exceed twenty minutes in the presentation of his paper, and no one shall speak longer than five minutes in the discussion of a paper. Members must send the titles and abstracts of their papers to the Secretary at least six weeks before the annual meeting." The Council announces that Article VII will be strictly enforced, and that titles and abstracts received by the Secretary after April first cannot appear on the program. The annual dinner of the Association will be held on the evening of May 12th. The Council recommends the following candidates for election to active membership. Dr. D. I. Wolfenstein, Cincinnati; Dr. J. M. Mosher, Albany; Dr. Harvey Cushing, Baltimore. The Council announces that in view of the increasing expenses of the Association, the dues for 1903 are ten dollars.

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Vol. 30 May, 1903 No. 5

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The Official Organ of

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The New York Neurological Society
The Philadelphia Neurological Society and
The Chicago Neurological Society

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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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# THE
# Journal
# OF
# Nervous and Mental Disease

## Original Articles.

### THE GEOGRAPHICAL DISTRIBUTION OF INSANITY IN THE UNITED STATES.*

BY WILLIAM A. WHITE, M.D.,
FIRST ASSISTANT PHYSICIAN, BINGHAMTON STATE HOSPITAL, BINGHAMTON, N. Y.

When I was invited by your Society to address you on the geographical distribution of insanity in the United States my ideas on the subject were extremely chaotic. I had vague notions of the possibility of formulating laws that would express the relationship between insanity and latitude and longitude, temperature, precipitation, etc., and I felt that a diligent study of statistics would be rewarded by the emergence of such laws. Similar ideas, I think, would quite naturally occur to any scientific man not especially acquainted with the statistical study of sociological phenomena. Confronted at the outset by the fact that the proportion of insanity varies greatly in different regions of the United States, what more natural than to ascribe such variations directly to the differences in man's physical environment in these localities?

From time immemorial variations in climate and in weather conditions have been supposed to produce profound effects upon man's conduct, and such expressions as the "depressing effects of heat" and the "stimulating effects of cold" are common in our every day conversation, and I believe that all of us have a more or less clearly defined idea that the physical and mental characteristics of the different races of men are to some extent an expression of the effects of the climatic and geographic conditions under which they live. This general conception was particularly fathered by that great English historian, Henry Thomas Buckle, who, in the opening chapters of his "History of Civilization in England" traces in detail the effects of the four great physical factors—climate, food, soil, and the general aspect of nature upon the characters of individuals and upon the growth of races and the progress of civilization.

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*Read before the National Geographic Society, Washington, D. C., Feb. 6, 1903.

There has consequently been fostered a general tendency on the part of statisticians and those engaged in the study of abnormal mental conditions, to follow along these lines with a view to establishing relations of cause and effect.

If I, in coming before you tonight, am not able to present to you such laws as I originally dreamed of, clothed in all the beauty of mathematical formulæ and demonstrating beyond doubt the precise effects of each climatic and geographic factor upon the prevalence of mental disease, I at least hope to be able to show why it not possible to do so, and I feel assured that my results may be just as valuable as if it were.

The social organism is extremely complex and any effort to reason from the association of two or more conditions to the probable causative relations between them is always dangerous, and when figures are suborned for such purposes the results are notoriously inaccurate. With the elaborate means used of late years by the governments of all civilized nations for the collection of statistics it is but natural that the figures obtained should be applied to all sorts of social conditions, and thus we are treated by the authorities to elaborate tables which show the month, day, and hour when suicide is most prevalent in a certain country, the season of the year in which crimes of violence reach their maximum, the effects of temperature, barometric pressure, humidity, wind velocity and precipitation upon various phases of conduct such as attendance at school, infractions of discipline in prisons, clerical errors in banks, etc., etc.

In view of these facts it is my function to-night to inquire whether the prevalence of insanity in the various regions of the

United States can be shown to have any definite relation to any one or more of these environmental conditions; whether insanity is more prevalent at certain elevations above sea level, or between certain degrees of latitude; whether it prevails more especially in regions of a certain average temperature and barometric pressure, or, on the other hand where the mean humidity is high or low, and further, if these conditions cannot be shown to have a causative effect upon its distribution—what has?

Let us start our inquiry by a study of a map of the United States, upon each State and Territory of which the ratio of insane to 100,000 population is indicated in accordance with the census returns for 1880. We are at once confronted with a condition of affairs which is so well marked that when I first saw it I was very much surprised. The greatest proportion of insanity is in the northeast, in the New England and Middle States, of which New Hampshire, Vermont, Massachusetts, Connecticut and New York all have one insane person to less than 400 of the population. If from this center of greatest prevalence of insanity we draw a line in any direction—west, south, or southwest—we see that no matter which way we go we find a steady decrease until we strike the Pacific slope. A slight interruption in the continuity of the decrease is noted in Michigan as we go west, but is, I think, of little consequence. As we go south along the coast Delaware appears as a marked exception. This is due to the fact that previous to the organization of the Delaware State Hospital in 1889, no statistics of insanity were reliable. The insane were county charges and the care given them was so atrociously bad that every one took pains to conceal cases occurring in their families. Despite these minor variations the decrease of insanity as we go from the northeastern part of the United States, south, west, or southwest, must strike you as being remarkably uniform and constant. This uniform decrease only takes place if we start from this northeastern center. If, for instance, we drop a line from any of the northwestern States, as Idaho, Montana or Minnesota, we find no uniform results, and if we go south from the Dakotas we will find that the proportion of the insane actually increases. The notable increase when we strike the Pacific slope I will speak of later.

If we now attempt to explain this condition of affairs by the topographical or the climatic conditions we are at once met by insuperable difficulties. If variation in temperature is alone responsible, why does not the proportion of insane diminish as we go south from the Dakotas as well as from the New England States? Or, on the other hand, why should Maine have a smaller proportion of insane than any other New England State? Montana, which is as far north as Maine, has a higher ratio than the States immediately south of it. If meteorological conditions are determining factors why do we not find a marked variation in the proportion of the insane in the States bordering on the Great Lakes? Here we have conditions quite different from anywhere else in the United States. This region, a large area of which is occupied by these immense inland seas, is directly in the course of the greater proportion of storms which come from the northwest and pass through here on their way to the Atlantic coast; sudden variation in temperature, barometric pressure and wind velocity are the rule and with the immense areas of evaporation, fogs and rains are frequent and the percentage of cloudiness unusually high (66%), still there is nothing in the proportion of the insane to call our special attention to this region.

I might continue in this wise, but it is only necessary for me to call your attention to the general results of such reasoning. They are these. The variation in the proportion of insanity in the different States is regular and uniform while both geographic and climatic conditions are not, but, on the contrary, differ greatly in different parts of the United States, as, for instance, in the region of the Great Lakes just mentioned. If, therefore, we would explain these figures we must seek a cause as uniform as its effects. This cause, or more properly, these causes, are the same causes that make for civilization, the same that make for permanency and organization of social institutions, the same that make for concentration of population in great cities, the same in short that make for progress in its broadest sense.

Before proceeding to the elucidation of this proposition let us for a moment return to the consideration of some first principles.

I did not intend to convey the idea, by the remarks I just made about the influence of climate on conduct, that no such influence could be demonstrated. On the contrary I think it can be and in fact has been. Dexter[1] has recently shown this in a most admirable and exhaustive study of the effects of climate on different phases of conduct. For instance, his studies show that as humidity increases assaults, necessity for prison discipline and the number of arrests for insanity decrease, while the data also show an increase in these same occurrences when the barometer is low.

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[1] Edwin Grant Dexter, A.M., "Conduct and the Weather," Psych. Rev., Vol. II., No. 10, May, 1899.

Granting for the nonce that these various meteorological conditions could actually produce insanity, they could not account for the uniform variation of the proportion of the insane in the different States to which I have called your attention. Weather changes are transitory and conditions that are inimical to mental health are quickly followed by others that are highly beneficial. This is especially true of those regions of the United States where the proportion of insanity is high. The ratio of insane in the semi-tropical regions, which are relatively free from the sudden changes of weather so common in the northeastern and northern central regions, is comparatively low. If we turn to the seasonal influences the same criticism applies, though the changes take a somewhat longer time. As regards climate and seasons, Berkley,[2] an eminent American authority, says, "These are factors of very minor importance in the evolution of insanity. The harmful effects of heat in the south are more than counterbalanced by the more prevalent abuse of alcohol in colder regions. In a general insane asylum, where the middle and lower classes of the population are received, a study of the records show that a larger number of admissions in one year may occur during the winter, whereas, in other years the same holds good for the spring, summer or autumn. Hence, one is obliged to conclude that the seasons have little to do with the evolution of insanity."

In the last analysis, however, the effects of all these agents, which collectively I have spoken of as constituting man's physical environment, upon his mind must be only secondary, mediate and not immediate. If we will study the effects of any one of them, for example, temperature, humidity, altitude, we will find them expressed in terms of respiration, pulse rate, evaporation from the cutaneous surface, blood pressure, etc. Effects which I grant you are potent, but which nevertheless are not primarily mental.

This whole matter reminds me very forcibly of the learned judge who could not understand why the expert called upon to testify as to the mental condition of the defendant, should have measured his feet. The medical profession has been largely responsible for this conception, especially our misguided friend the gynecologist. This gentleman has insisted that all forms whatsoever of mental disease affecting the female were traceable to an affection of the uterus or its appendages, and has devised all manner of operations to relieve such conditions. True, the insane female who may have a local pelvic condition which is amenable to surgical interference is just as much entitled to the relief that can be obtained from that source as her more fortunate sister, and it is quite conceivable that the relief of a local condition which was painful or debilitating by reason of frequent hemorrhages, or other cause, would place the organism in a better condition to rally from any abnormal state. But the sort of stuff that mind is made of is not to be found in the abdominal cavity.

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[2] Henry J. Berkley, "Insanity: General Etiology," Reference Handbook of the Medical Sciences, Vol. V.

This brings us again to the basis of our argument. If we are to seek for adequate causes to explain the conditions to which I have directed your attention we must seek for mental causes, not physical ones.

If we look back over organic nature we shall see that in the progress of evolution the nervous system has come to play a progressively more and more important part until we get to the higher animals, the vertebrates, in which the brain comes to be of paramount importance.

Still, in the lower races of man, although the brain is of such great importance in the struggle for existence, that struggle is, after all, in the main and relatively a physical struggle. It consists largely of collecting food which is often ready at hand in the tropics, of pursuing and killing game, and often of personal encounters with his fellowman, as a result of which the conquered is killed or reduced to slavery. When we get to civilized man, however, the picture is different. Here the struggle for existence has become an essentially mental struggle and success is a function of intellectual capacity. I can in no better way illustrate the severity of this struggle than by calling your attention to the fact that it takes twenty-five years of preparation now-a-days before a young man is considered equipped to cope with his fellows.

The brain then becomes, as it were, the storm center in the organism. Here, in the habitation of the mind, do all the problems of subsistence meet their solution, and here also do all those mighty emotions which ever and anon stir the soul, take their origin. It is here in the brain that vaunted ambition has its sway, and here that the sweet pains of love tune one soul in harmony with another.

The mind, delicately adjusted as it is to its environment, responding as it does to the slightest changes therein, occupies a dangerous position and becomes at once liable to great stress and to the multiplicity of disorders that result therefrom. The savage in his simplicity does not know what it is to suffer from the cares and worries which are the daily portion of the average European, and it is little wonder that the latter, beset by all manner of disappointments and vexations, should more frequently break down in mind than his less gifted brother.

If you have followed me thus far you will note that in my attempt to account for the geographical distribution of insanity in the United States I have discarded the influences of the physical environment as being efficient causes because of their indirectness, and have appealed to the immediate results of mental stress, the results of the contact of man with man in the struggle for existence; in short, the results of that struggle itself as exemplified in civilization.

If my contention is true that insanity is the result of the stresses incident to the progressive civilized state, it must be possible to educe further proof of this by a study of some of the phenomena that accompany civilization. We would thus expect to find that in those localities where civilization was furthest advanced, where the social institutions were stable, where class distinctions had crystallized, in short, where the stresses of intellectual life were greatest, the proportion of insanity was highest. Let us see if this is so.

One of the most marked results of civilization is the concentration of population in certain areas. Let us study this condition in the United States with reference to the distribution of insanity. The census for 1890 shows that for the different regions of the United States the population per square mile is as follows: North Atlantic Division, 107.37; South Atlantic Division, 32.98; North Central Division, 29.68; South Central Division, 18.94; Western Division, 2.58. The North Atlantic Division, comprising the New England States, with New York, New Jersey and Pennsylvania, has more than three times the number of inhabitants per square mile of any of the other divisions; in fact, more than all the rest put together. Of these States Rhode Island, the smallest, has the greatest density of population, with 318.44 to the square mile; then comes Massachusetts with 278.48, Connecticut with 154.03, and down the coast, New York with 125.95, New Jersey with 193.82, and Pennsylvania with 116.88. From this center of density the proportion of inhabitants to the square mile diminishes regularly in every direction. If we go south we find Maryland with 105 and Delaware with 86 per square mile, until in the extreme south we find but 30 or 40. Westward from Pennsylvania, however, we find a belt bordering the Ohio river, containing Ohio with 90, Indiana with 61 and Illinois with 68 per square mile, and from here the diminution is rapid to Louisiana with but 24, Minnesota with only 16 and the extreme west, where the proportion is less than one.

Here you see we have an almost exact parallel with the distribution of insanity.

Closely connected with this peculiarity of civilized communities to concentrate in certain areas, in fact, a part of the same phenomena, is the growth of great cities. The eleventh census shows that the percentage of the population of the United States living in cities of 8,000 or more inhabitants, for the different regions were as follows: North Atlantic Division 51.58; North Central Division, 25.91; South Atlantic Division, 16.03; South Central Division, 10.45; Western Division, 29.99. Here again we see the same parallelism between the degree of manifestation of a phenomenon of civilization and the proportion of insanity. The North Atlantic Division contains almost twice the percentage of urban population of any of the other divisions, and here as we know we find the highest percentage of insane.

If we calculate the proportion of insane per 100,000 in all cities of the United States containing 50,000 or more inhabitants we will find that in 1880 the ratio was 231.6, as against 183.3 for the whole country, while in 1890 the ratio was 242.7, as against 170 for the whole country. Thus we find that the ratio of insane in cities of this size has not only increased in the decade from

1880 to 1890, but that the ratio for the whole country has decreased. It is also significant that, while in 1880 there were only 35 cities containing 50,000 or more inhabitants, in 1890 there were 58 such cities. We further find that of these 58 cities 26, or nearly one-half, are located in the North Atlantic Division. Of these twenty-six 6 are in Massachusetts, 7 in New York, 5 in New Jersey, 5 in Pennsylvania, 2 in Connecticut, 1 in Rhode Island, and none in Maine, New Hampshire and Vermont, so that our findings thus far are still further harmonized by these additional facts, for the density of population in Maine, New Hampshire and Vermont is very much lower than for the other States in this region..

If now we study the movement of population during the past century we meet again the same confirmation for our views. Mayo-Smith[8] makes the statement that in 1790, 95 per cent. of the population were on the Atlantic seaboard, with an average depth of settlement at right angles to the coast of only 255 miles. The stream of population spread westward along three lines, one the valley of the Mohawk, one from Virginia southwest into Kentucky and Tennessee by way of the Appalachian Valley, and one over the Alleghenies to the Ohio river. This latter course was the principal one, and from the junction of the Ohio with the Mississippi we find further westward migration occurring along the valleys of the Missouri, Arkansas and Red rivers. This course of the westward spread of population has been maintained, for, though valleys furnish the natural highways for migration, when railroads come to be built they are built in the valleys and the general course of events is not materially changed thereby.

See how closely these facts correspond with the distribution of insanity. From the northeastern coast States—Massachusetts, Connecticut, New York and New Jersey—there is a progressive decrease southward along the Atlantic coast. There is also a decrease as we go southwest along the Appalachian valley, but we find the ratio of insane continues high in the Virginias, Kentucky, Tennessee and North Carolina, and does not show a marked falling off until we get south of these States. Similarly, if we follow the Ohio valley we find the ratio of insane continues large in Ohio, Indiana and Illinois.

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[8] Richmond Mayo-Smith, "Statistics and Sociology."

We still have, however, some high ratios unaccounted for, viz., Missouri and Iowa west, Michigan, Wisconsin and Minnesota north. All these States are in the North Central Division. Let us compare the different divisions of the United States on the basis of their respective increases in population during the decade from 1880 to 1890. The figures are as follows: North Central Division, 4,878,928; North Atlantic Division, 2,984,480; South Central Division, 1,985,657; South Atlantic Division, 1,204,999; Western Division, 1,129,641. Thus we see that the increase in population has been by far the most rapid in the North Central Division, this territory has increased approximately 2,000,000 inhabitants more than any other. Let us now turn to the individual States and see what the figures show. The States in the North Central Division which have increased in population the most are in the order of their increase: Illinois, 747,629; Nebraska, 603,399; Minnesota, 519,069; Missouri, 510,262; Ohio, 473,856; Michigan, 451,170; Kansas, 430,167; Wisconsin, 367,420; Iowa, 287,156. The only other States in the Union that have increased at any such rate as this are New York, 911,173; New Jersey, 313,103; Pennsylvania, 972,962; Massachusetts, 454,432; Texas, 642,357; Arkansas, 325,344; Georgia, 294,992; Washington, 266,999, and Oregon, 258,300.

The significance of these figures seems to me quite evident; they show that the stream of population has continued west of the Mississippi, and the high rate of insanity in Iowa and Missouri is therefore accounted for, as these States both adjoin Illinois, the western limit of the Ohio valley lying merely on the other side of the Mississippi river. You will see also that we have incidentally thrown light on the high ratios north. In the three States in this region, Michigan, Wisconsin and Minnesota, there has been an increase in population in the ten years from 1880 to 1890, of one and one-third millions.

Of all these States Nebraska alone seems to be somewhat exceptional. Although its population has increased rapidly its ratio of insanity is rather lower than we would expect from comparing it with those States where the increase has been correspondingly marked. Of these States Kansas is the only one as far west as Nebraska, and Kansas has a ratio of 125.7 per 100,000, while Nebraska has but 88. It is significant in this connection that Kansas is more directly in the line of traffic from east to west, and a glance at any recent map of the United States will show that many more railroads course through it than do through Nebraska. As both of these States are in the main agricultural, the higher ratio of insanity in Kansas would seem to me to be the result of the degenerate dribble from the great railroad lines as they pass west from the congested centers of population in the east.

The only reports of the railway mileage in these two States I have been able to obtain are one under date of 1893, which shows Kansas to have 8,900 miles of railroads, thus making it the second State in the Union in this respect, and one a year later in 1894, shows Nebraska to have but 5529.22 miles of railroads.

It would seem, therefore, that my contention that insanity increases in proportion as the stresses incident to the struggle for existence become mental stresses, is borne out by the facts. The frontiersman who takes his family and goes west to open up new territory, engage in legitimate agricultural pursuits, and grow up with the country, is pretty apt to be of hardy stock, and insanity, if it appears at all, comes in later generations. It is different, however, with those States that have great mineral wealth. Here the attraction appeals to all the wandering, unsettled rifraff of the country, who hasten to the newly discovered fields in the hope of acquiring a fortune quickly. Arrived there they yield to all the seductions of intemperance; vice and disease wreak their ravages upon a predisposed soil, and our ratios show a corresponding increase. This is the situation with California. This State, and to a somewhat less extent, the whole Pacific coast, is still suffering from the effects of the "gold fever" of '49, and its citizens are paying the price even "unto the third and fourth generations." In this connection it is interesting to note that the mining States and the States of the Pacific slope, viz., Montana, Colorado, Arizona, Nevada, Idaho, Washington, Oregon and California, all show a much greater number of male than female insane, a condition that prevails nowhere else in the country, with the single exception of Minnesota, and it has arisen here almost wholly in the decade from 1880 to 1890, during which period the State has increased in population over half a million. Minnesota also has large lumbering interests, and conditions in a lumbering region are similar to those in a mining region. In the normal order of things we expect to find a slightly higher percentage of insanity in the female sex, but the "get-rich-quick" fever attracts more men than women, and mining districts as a rule are deficient in their proportion of women. This state of affairs has apparently not yet been recovered from in California. We must also remember with reference to California in particular that it is a coast State and suffers from the effects of immigration, and that the percentage of insanity is invariably higher in the foreign born than in the native population.

This law of the increase of insanity in the oldest settled districts and its decrease in the newly settled districts was well stated by A. O. Wright in the *Proceedings of the National Conference of Charities and Correction,* in 1884. He said: "A very powerful cause for the increase of insanity in this country was, so far as I know, first pointed out by the writer in 1881, before the census of 1880 had been tabulated, in the Annual Report of the Wisconsin State Board of Charities and Reform, and was stated in debate at the National Conference of Charities and Correction at Madison in 1882. Having made a census of the insane under public care in Wisconsin, the writer, on reducing the number by counties to the ratio to the population of the several counties, was astonished to find here a general law: That the older settled counties had the largest ratio of insane to the population, and that the ratio steadily decreased and reached the smallest ratio in the pioneer counties on the north. This seemed to show that a new country has a smaller proportion of insanity than an old country.

"When the Compendium of the Census of 1880 was published, the writer, from the numbers then given, immediately calculated the ratios to the population and arranged the States and Territories geographically instead of alphabetically." From the figures thus obtained he concludes that " . . . . allowing for exceptional cases, the proportion of insanity decreases as you go toward the newer settled States, from about one in every 350 of the population in Massachusetts to about one in 1900 in Colorado."

Wright, however, does not go into details nor discuss the causes that have led to this condition of affairs, except to say: "The reason of this I think to be that new settlements are made by a selected population, mostly young and middle-aged people of sound minds and bodies. The insane are left behind, as are also those people of bad organizations from whose numbers the most of the insane will come. The new countries, therefore, have a small proportion of insanity at the start, and furnish a small proportion of insanity in the first generation.

"The only exception to this is in the case of the Pacific slope and a few other localities, where masses of homeless men, with few women and children, have gone in search of work or of wealth; where the vices of drunkenness and licentiousness, with the irregularities and the hardships of life in mining or lumbering camps, and the excessive fluctuations of fortune, have caused an excess of insanity. In these cases, it is, however, to be remembered that this is a disease of mature life; and, if we add the proper proportion of children who would be found in an ordinary community, and who rarely have insanity, we should at once halve the ratio of insanity in such communities.

"But, in ordinary settlements, where the settlers found homes, and live under the ordinary conditions of life, the ratio of insanity in the first generation is small, because they are, as the insurance men would say, 'selected lives.' In the second generation, all the complex and varied causes which produce insanity have been at work; and the second generation has a much greater ratio of insanity than the first, and so on for several generations, when the balance is restored, and the regular rate of insanity is reached."

After all this, however, Wright says: "It is often claimed that insanity is a disease of civilization, and that it is increasing because civilization is increasing. This I think to be a mistake." Although this is not a very happy way to express it, it seems to me that our figures prove just that, or, rather, if they do not prove that insanity is the necessary result of civilization, they at least prove that the civilized state offers those conditions in greater number which bring it about, and so if the connection be not one of necessity it is at least one of fact. Instead, therefore, of attempting to account for insanity by altitude, temperature and the various other elements of the physical environment, we should only consider these factors as important because of their influence in creating conditions favorable to the growth and concentration of population and the evolution of the social organism. Even here this influence is often secondary, or accidental. As regards this whole matter of the influence of the physical environment on population I can do no better than quote Mayo-Smith,[4] who, in answer to the question, "How far can the statistics of distribution be said to contribute an answer to the question of the influence of physical environment upon population?" says:

"Statistics show us, in a large way and on a grand scale, the general influence of land, climate, and natural forces upon population. The plains attract, the mountains repel. Cold regions are unpopulated; moist and warm climates are fatal to human life. Commercial position attracts cities; navigable rivers are natural highways and are utilized in the migrations of the human race; an indented sea-coast is favorable to settlement and colonization. Statistics confirm the general observations of history. Levasseur, after a long survey of the topography of France and the history of its population, says that at all periods Paris has been the attractive pole, and the mountainous region of South France the repulsive pole of population.

"But it is absurd to seek by statistics a direct mathematical relation between population and land. The population of a country is not dense exactly in accordance with its topography. Plains do not always have a dense population, and mountains are not always barren. Population does not increase or decrease regularly, according to distance from a certain parallel of latitude or longitude. There is no direct proportion between the degrees of temperature or inches of rainfall, and the number of inhabitants in a certain district. In this respect many of the statistics distributing population to topographical features or natural relations, such as those of the Tenth and Eleventh Census of the United States, are the merest vanity. One searches in vain in these elaborate tables for any illumination. Such influences are not direct, but indirect. Altitude, temperature, rainfall, influence population because they affect the economic resources necessary for population. We must always remember that economy is the basis of social organization. The economic is the fundamental side of civilization. Natural forces control human life in this way. Statistics, by showing the distribution of population, disclose the harmony between population and nature which is mediated by economic relations; and these are, on the one side, the result of natural forces, and on the other, the conditions of human existence.

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[4] Ibid.

"We must also remember, in studying the distribution of population, that there are commonly many influences at work, some of them economic, others historical and political; and that it is often extremely difficult to disentangle them. We ought, therefore, to expect from statistics, not exact data, but only general indications of the influence of natural forces. The density of population in England, for example, is due partly to the richness of its soil, partly to its mineral resources, and partly to its commercial advantages; but it is due also in part to its insular position, which has given it peace and stable government for generations, and to the energy and enterprise of its inhabitants, which have made the little island the center of a world empire. It is impossible for statistics to disentangle these different influences. It can only confirm the observations of history. Who could explain that oasis of population, in the great western plain of the United States, called Utah, if he did not know the history of the Mormons? Why should the sterile mountain-tops of Nevada be populated? might be asked by one who did not know the history of gold and silver mining. The coast swamps of the United States would probably be uninhabited did not the population of the United States include a large proportion of negroes, who are proof against pestilential fevers. Race explains, in this case, what physical geography would leave inexplicable.

"Finally we must remember that all these natural influences are much more powerful over primitive than over civilized man. As Spencer says, 'The earlier stages of social evolution are far more dependent on local conditions than the later stages. Those societies such as we are most familiar with, highly organized, rich in appliances, advanced in knowledge, can, by the help of various artifices, thrive in unfavorable habitats; yet feeble, unorganized societies cannot do so: they are at the mercy of their natural surroundings.' Spencer finds here, also, the explanation of the fact that so many tribes of savages have made no manifest progress during the long period over which human records extend. Statistics observe man only in an advanced state of civilization, when he has been able to free himself to a certain extent from the influence of natural forces, or at least to neutralize them. By clothing and improved shelter man habituates himself to almost any climate, and by sanitary knowledge he makes places formerly uninhabitable safe for human life. In pursuit of wealth, of political independence, of religious freedom, he will risk exposures which would seem to be entirely unnecessary. By improved methods of agriculture man often renders districts, formerly uninhabited, or at best only sparsely settled, capable of sustaining large populations. In early times regions covered with forests are thinly inhabited. Civilized man cuts down the forest and turns the land into arable fields. Lowlands, which in early times were at the mercy of the sea or uninhabitable on account of fevers, civilized man, by canals and dikes, renders fertile plains. So also, by means of fertilizers, by rotation of crops, by improved ploughing, by the use of machinery, sometimes by irrigation, dry and sterile plains are made productive. Even from year to year changes in agriculture, or in the prices of agricultural crops, may render it expedient to change arable land into pasture, or pasture land into arable; and either process, if continued, must influence the population-supporting capacity of the country. An example of this is seen in the changing of arable land to pasture in Ireland, and the turning of cattle farms into game preserves in Scotland.

"In the civilized state man often makes use of a country without any reference to its agricultural capacities. He seeks the minerals under the soil, either for his own consumption or for export; he turns clay into pottery; he utilizes water power for his factories; he seeks barren coasts for fishing or gathering seaweed; he establishes trading posts in the desert, or in unhealthy localities; in other words, he seeks his gain without reference to climate or soil. In modern times the improved means of transportation have still further increased man's command over nature. He is no longer held to rivers and valleys as natural highways, but can seek the quickest and most direct route. Cheapness of transportation gives him command over the resources of the world. In this way he can carry on the work of production in any place he likes, without regard to its food-producing capacity. The people of England import three-fourths of the bread they eat. This has the effect of enabling man to concentrate his efforts in places most favorable to the production of the kind of wealth which is demanded. It enables him also to choose climates favorable to his health, as the English seek the Mediterranean, or consumptives of the east seek the dry air of Colorado. Man's intellectual and emotional desires lead him to seek large cities, and this he is enabled to do by the fact that he can carry on his occupation independent of the food supply. This is especially true of occupations demanding intellectual effort.

"It will be seen, therefore, from all these considerations, that man is still subject to the environment, but the development of his power over nature has rendered the cord which binds him down more elastic. He is still subject to nature, but has at the same time, to a certain extent at least, subjected her."

Thus far my discourse has dealt with:

1. The untenability of any hypothesis founded solely upon climatic, meteorologic, or topographic conditions to explain the facts of the distribution of insanity in the United States.

2. The necessity of assuming primarily a mental cause to explain these facts and the nature of that cause, viz., the mental stresses incident to the progressive civilized state.

3. Now as a third line of argument I will take up the discussion of certain collateral evidence, that is, evidence taken along other but related lines and leading to the same conclusion.

Suppose we first examine into the statistics of suicide. Morselli, in his admirable work on that subject, comes to the conclusion that those sections of Europe show the highest percentage of suicide where the Teutonic element is predominant. Ripley, in his excellent work, "The Races of Europe," has examined this proposition critically and with very interesting results.

If, for instance, France is studied, we will find the greater proportion of suicide in the north, where the Germanic race is represented in greatest numbers. Similarly we find here also the highest divorce rate. But more remarkable still, we find evidences of the highest degree of culture. In this same region the greatest number of artists were born to whom were granted awards by the Paris Salon, and here also were born the highest ratio of men of letters. If now Italy be similarly studied we find that its different regions are distinguished in much the same way as they are in France, by a preponderance of certain phenomena in certain localities. In comparing the two countries Ripley closes his criticism by saying, "The effect has been to emphasize once more the enormous preponderance of artistic genius all through the north, from Tuscany to the Alps. How does this coincide with our previous deduction concerning France? It seems, perhaps, to corroborate the relation of Teutonism to art, until we secure the fact that all northern Italy is overwhelmingly Alpine by race as compared with the artistically sterile south. Couple with this the fact that in reality Teutonism is a negligible factor in Italy, physically speaking, and that precisely the same ethnic type which is so fecund culturally in Italy, is in France, the one localized wherever art is not; and all doubt as to the predominant cause of the phenomenon is dissipated. We see immediately that the artistic fruitfulness in either case is the concomitant and derivative product of a highly developed center of population. Contact of mind with mind is the real cause of the phenomenon. It is not race but the physical and social environment which must be taken into account."

Morselli himself recognized this fact for he not only reaches the conclusion that "It is those countries which possess a higher standard of general culture which furnish the largest contingent of voluntary deaths," and "The proportion of suicides in all Europe is greater amongst the condensed population of urban centers than amongst the more scattered inhabitants of the country." But in concluding his work he sums up the whole matter in the following words: " . . . whoever has followed us in the long analytical course which we have pursued, ought now to be convinced of the connection between competition and social evolution, and the inclination towards suicide. Suicide increases amongst people according to their degree of civilization, not so much because in the high development of the cerebral organism the needs which must be satisfied increase, as because the brain shares more largely in the struggle."

I need only call your attention to the frequent association of suicide with actual insanity, or at least with an abnormal mental condition for you to see the bearing of these results on the problem in hand.

Pauperism is another allied condition to which I would direct your attention. The census of 1880 shows that there were then 66,203 paupers in the several almshouses of the country. Now pauperism is to an extent a symptom of mental defect. The individual who, unless absolutely incapacitated by physical disability, so far fails in the struggle for existence that he must be supported at the public expense, is certainly suffering from some form of mental defect. F. H. Wines, the special agent of the census office for the collection of the statistics of the defective, dependent and delinquent classes at the tenth census, says about pauperism, "The law which governs the distribution of pauperism in the United States (and which, we believe, has not been suspected by any student of the subject—at least I have never seen any reference to it) is brought out as clearly by the census of 1850 as by that of 1880, and it is confirmed by every census that has been taken. This law is as follows: The ratio of paupers to the total population diminishes alike from north to south and from east to west. In other words, if New England, or the principal New England State (Massachusetts), be taken as a starting point, it matters not in which direction a line be drawn, the largest amount of pauperism, relatively to the population, will be found to exist in Massachusetts, and the smallest in the State farthest removed from Massachusetts; while the intervening States will exhibit, on the whole, and with scarcely an exception, a gradual decline in something like the degree of their removal from the extreme northeast." As clearly as Mr. Wines defines this law it is rather strange that he did not discover the practically identical condition relative to the insane.

We have one other state of affairs in the United States that is worth while looking into. I refer to our large negro population. The ratio of insanity in the negro population is smaller than in the white population, being as 1 to 1,069 in the former, and 1 to 505 in the latter. (Census 1880.) Although this is so it is generally admitted that the percentage of insanity has been gradually increasing since the Civil War. Berkeley[5] says on this point, "Before the Civil War there were few or no psychoses among them, and such organic degenerative diseases as syphilitic insanity and dementia paralytica were practically unknown. Today in communities where many are collected, as in Washington or Baltimore, the percentage of insane negroes, not to mention idiots and imbeciles, is already fully up to that of the Caucasian races with whom they are associated, and bids fair to surpass it.

"The negro has been thrown upon his own physical and mental resources and has entered the strife for existence as an inferior; he is syphilized, alcoholized, his food is ofttimes unsuitable . . . his surroundings are usually unhygienic, and tuberculosis finds in him an easy prey. No wonder is it that under these circumstances we have in our asylums an ever-increasing number of idiots, of imbeciles, and of all types of the dementias from the colored race."[6]

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[5] Ibid.

There are, however, some extremely interesting facts relative to this increase. The percentage of colored insane increases rapidly as we leave the natural home of the negro and go in any direction. In other words, as soon as the negro goes north and enters into active competition with the white, who is mentally his superior, he succumbs in the unequal struggle. So in Georgia where we find the greatest number of negroes, there was one insane negro to 1,764 of the colored population in 1880, while in New York the ration was 1 to 333, or almost exactly the same ratio as for the white population.

Then again if we take the southern States alone, viz., Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, Tennessee, Texas and Virginia, we find the ratio of colored insane is 1 to 1,277, while for the whites in the same territory it is 1 to 456. For the remainder of the United States the ration of colored insane as shown by the tenth census was 1 to 542, while for the whites it was 1 to 520. The ratio of colored insane in the United States minus the southern States, is then almost exactly the same as the ratio for the white insane.

It seems that all the lines of evidence I have followed up lead to the same conclusion; they are mutually confirmatory of the general law that the proportion of insane is highest where we find the greatest congestion of population, and therefore, where the stresses incident to active competition are most severe. Our inquiry thus far, however, has been nothing if it has not been an inquiry into the causes of insanity, and I think I may fitly close by a general discussion of causes with a view to indicating some general conclusions relative to the comparative influence of these mental stresses I have been discussing in the actual production of insanity.

If we will take up any annual report of an institution for the insane and turn to the table giving the causes of insanity in the several patients under treatment we will find assigned such causes as these: "business anxiety," "death of mother," "disappointment in love," "domestic troubles," "excessive study," "loss of property," "political excitement." How many of us but have suffered at some time or other from one or perhaps all of these so-called causes of insanity? Certainly we have all had business worries; certainly we have all lost property at some time, otherwise our good fortune is phenomenal; certainly we have all been subject to political excitement many times, and all of us presumably have lost a dear friend or relative, perhaps a father or mother. Dr. Carlos F. MacDonald says very forcibly on this subject: " . . . that substantially every individual at some time during his life is exposed, in many cases repeatedly, to many of the so-called exciting causes of insanity, both mental and physical, and yet, despite this fact, we find that sanity is the rule,—insanity, the exception."

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[6] "A Treatise on Mental Diseases."

In ascribing these causes what has been done is simply this—the particular set of conditions that happened to maintain at the time the patient was attacked with insanity has been tabulated as the cause of that attack, whereas the true cause was in all probability far removed from these which were in reality only accidental contemporaries. In reality the true underlying condition in all these cases for which such causes are assigned, is the predisposition to insanity.

Predisposition to insanity may be either inherited or acquired. The former is more generally recognized and is what is referred to when insanity is said to be hereditary. Of all causes of insanity heredity is recognized as being by far the most important and as being most frequently present. The average for all countries has been estimated at from 60 to 70 per cent. This, I believe, as a matter of fact, falls below the truth, But any one who is at all familiar with the collecting of statistics must know how impossible it is for them to fully represent the facts in such a matter.

Next to hereditary predisposition comes acquired predisposition as a factor in causation, and the two most important agents in bringing about this acquired predisposition are generally acknowledged to be, first, alcohol, and second, syphilis, both of which, however, may act as true exciting causes at times. It is further conceded that both of these causes are much more prevalent in civilized communities, and in fact seem to be fostered by that irregular life which the active struggle after wealth necessitates.

The inadequacy of predisposition alone to account for insanity, especially acquired predisposition due to alcohol, syphilis, and tuberculosis without the element of mental stress, is well illustrated by the condition of the American Indian. Sorely afflicted as he is by the diseases and vices of civilization, his tendency is to an outdoor life, and as his land has disappeared and he has become physically incapacitated, the Government has supported him, so that his sufferings have been in the main physical and not mental. Careless, slovenly and improvident, he does not know much of worry for the morrow, and so we find that among his race "insanity is of rare occurrence." [7]

Without wearying you with further figures I will simply call your attention to the new light in which our conclusions now appear. Insanity is most frequent in the older civilizations, in the more thickly settled communities, in urban centers, in short, where competition is most active. Here the weakling, the man whose mental faculties are not quite up to grade, who enters the struggle handicapped by a poorly equilibrated mind, goes to the wall. He is the victim of heredity. Here are bred all the vices which only a high grade of intelligence can call into being, stimulants, narcotics, drugs of all kinds are available to help the overburdened on their way, until at last they react and bring ruin and desolation. The victims who fall a prey to these temptations are the victims of an acquired predisposition.

Of these two varieties of causes heredity is by far the more important. While civilization furnishes the environment that makes a bad heredity doubly dangerous, still it is the heredity which is the prepotent factor and not the environment. A bad heritage is always a source of danger and its possessor can never know when the environmental conditions may appear which will make its latent activity kinetic.

No people in the world are freer than we are from the taints of vicious inheritance. Inhabitants of the most glorious country on earth, a country whose future for greatness, and power, and good, seems to have no limit, let us see that we make the best possible use of the bounties nature has showered upon us with so prodigal a hand.

But power and greatness are double-edged; they cut both ways, and already we are threatened with the dangers they have brought in their wake. The off-scourings of all Europe are hastening to our shores for that wealth they expect to find ready at hand, and today 50 per cent. of the nearly 25,000 insane of New York State are foreign born. The result of this great influx of defectives must of necessity have a constant leavening effect on the whole population. The danger from this source, however, is as nothing compared to that from war, the greatest curse that can afflict a nation.

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[7] "The Civilized Indian, His Physical Characteristics and Some of His Diseases," by A. D. Lake, M.D. Trans. N. Y. Med. Soc. 1902.

In war it is not the defective that goes down to death, but the flower of a nation's manhood, and if modern theories of heredity are correct their place can never be filled—once gone they are gone forever, while the maimed, the diseased, the imbeciles and degenerates, unable to sustain the hardships of campaigning, stay at home and help populate the country with their ilk. I believe one of the principal reasons for this country's prosperity lies in its freedom from foreign wars, and I am convinced that no more terrible calamity could happen to it than to be engaged in one.

If we can control these two sources of evil successfully I am sure that internal affairs will so shape themselves as not to seriously interfere with a future which I believe can today only be dimly imagined, a future which will outshine the glory of ancient Rome as good outshines evil.

# THE CHANGES FOUND IN THE CENTRAL NERVOUS SYSTEM IN A CASE OF RABIES WITH ACUTE MENTAL DISTURBANCE.[1]

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The pathological anatomy of rabies has been studied by numerous observers, both in the human subject, and in various animals, notably in the dog and in the rabbit. The results of all coincide in showing that the poison of the disease spends its force chiefly upon the nervous system, the changes described being hyperemia and dilatation of vessels, smaller or larger hemorrhages, round cell accumulation both in the sheath of the smaller vessels and in the perivascular spaces, degenerative changes in the nerve cells, local areas of softening, and meningitis in different portions of the central nervous system, but usually most marked in the bulb. The process according to Golgi is to be considered as an acute encephalo-myelitis. The part of the nervous system most affected has seemed in some cases to bear a relation to the location of the infecting bite, Schaffer having found profound changes in the anterior horn of the cord in the lumbar region of the corresponding side in a patient bitten upon the leg, while in another person bitten upon the arm, the cervical region of the same side was specially affected. The anatomical changes described have, however, been in the main such as are observed also in other diseases. Babes has called attention to the presence especially in the bulb, of collections of round cells grouped characteristically about the nerve cells of that region, to which he has given the name of "rabic tubercle," and to which he attaches great diagnostic importance. As far back as 1872, Pollailon and Nepveu in a case of rabies found the Gasserian ganglion infiltrated with round cells, and compressed, but this lesion does not seem to have been considered at that time of much importance. In 1899 and 1900 Van Gehuchten and Nélis, in a series of articles, called attention to the changes which they had observed in a number of cases of rabies, in the ganglia of both cranial and spinal nerves, and which they considered as specific. These changes consist in round cell invasion, and especially in the proliferation of the endothelial cells lining the capsules in which the large nerve cells of the ganglia are placed. This proliferation may go on to the complete destruction and disappearance of the nerve cells, the capsules being filled with round cells, presenting in some instances an appearance not unlike an alveolar sarcoma. These changes are said to be most characteristic in the dog, less so in man. The matter was speedily taken up by other observers, and in the dog and the rabbit at any rate the changes described have been thought to be characteristic. For their production the action of the fresh virus, and the complete evolution of the disease seem necessary, and Van Gehuchten and Nélis have considered them of diagnostic value only in street rabies. Sano examined the ganglia from a case of human rabies, but failed to find in them the changes described as characteristic by Van Gehuchten and Nélis, while Crocq, after a thorough study of the subject, is not convinced of the specificity of either the ganglion lesions or the rabic tubercle of Babes, though he admits that each may have very considerable diagnostic value in a suspicious case. In this country the subject has been specially studied by Ravenel and McCarthy, who found the changes described by Van Gehuchten and Nélis, in the ganglia of a number of dogs and rabbits, of one cow, and in one human being. Dr. McCarthy has, I believe, also reported some additional cases in man, though his paper has not yet appeared in print.

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[1] Read before the Philadelphia Neurological Society, February 24, 1903.

So far as I can ascertain no one else in this country has as yet published the results of an examination as to the presence or absence of the lesions claimed to be specific in a case of human rabies. The following case came under the observation of the writer during the past fall.

R. T., white, a farm laborer thirty-two years old, addicted to the use of alcohol, but to what extent not ascertainable, about August 15, 1902, being at the time intoxicated, was bitten on the hand by a dog which had been acting strangely, whose mouth he was attempting to open, as he explained, to find out whether the animal was mad or not. The dog in question was said to have been bitten by another dog which is stated to have been mad, and to have also bitten several other dogs and a cow. There is no record as to an examination of any of these animals, but the cow is reported to have remained well. The dog which bit R. T. was killed shortly afterward, and no examination of his body was made. The patient, R. T., having listened to numerous comments upon the occurrence, and having had the symptoms of rabies repeatedly detailed to him by his neighbors, grew nervous and depressed, gave up his work, and began to drink heavily. Upon November 8 he became excited and violent, tore his clothing, is said to have "barked like a dog," was unable to swallow, and took neither food nor drink from that time on. He was brought to the New Jersey State Hospital at about midday November 11, tied hand and foot. At this time he was extremely restless and excited, kept constantly in motion, secreted a great quantity of saliva, and was absolutely unable to swallow. He did not seem to have any definite delusions, hallucinations or illusions, and in an interval of comparative calm told the attendant that he had hydrophobia and hated to die.

On account of the questionable history, however, the case was regarded as being most probably one of acute excitement supervening upon alcoholism. Nevertheless, as the patient was in a condition of extreme exhaustion an unfavorable prognosis was given. This was speedily verified by his death at 9.30 the same evening. An autopsy was performed at 2.30 P.M. on November 12.

The macroscopic findings were: Adherence of the dura along the posterior part of the longitudinal fissure, with thickening of the pia-arachnoid in the same region, moderate congestion of the vessels of the meninges, and to a less extent of those of the brain, adherence of the lungs, and congestion of their bases, and congestion of the kidneys. The gastro-intestinal tract showed no special change. Portions of the brain and spinal cord were immediately forwarded to Dr. Ravenel at the Pepper Laboratory, with the request that he inoculate some rabbits for me. This he kindly did with the result that each of the two animals used developed typical paralysis of the hind limbs on November 31, and died on December 3, after the paralysis had ascended to the fore limbs.

There were taken for microscopical examination, portions of the cortex from the paracentral, frontal and occipital regions, and from the cerebellum, sections of the brain axis at different levels from the lower medulla up into the internal capsule, pieces of the spinal cord from different regions, the Gasserian ganglion, and several spinal ganglia, besides parts of the lung, heart, liver, spleen and kidney. The sections of the nervous system were stained by methylene and toluidin blue, by Held's method, by carmine, hematoxylin and eosin, the Wolters-Weigert, and the Marchi methods. Most of the large cells of the paracentral lobule, of the bulbar nuclei, of the anterior horns of the spinal cord, and of the columns of Clarke, stain diffusely, showing no granules. Many of them contain a deposit of dark pigment, and in a few there is displacement of the nucleus towards the periphery.

Throughout the whole nervous system, there is dilatation of small vessels and much round cell accumulation both in the perivascular spaces, and in the sheaths of the vessels, these changes being most marked in the brain axis, but also evident in the cortex, and in the cord. In various locations there are small hemorrhages, notably in the vagus and hypoglossal nuclei. A careful search for the rabic tubercle of Babes, shows in a few places a grouping of round cells about the nerve cells. This is plainest in the vagus nucleus, in one of the arcuate nuclei, in the external acoustic nucleus, among the deeply pigmented cells of the substantia nigra cruris, and in the optic thalamus.

In the Gasserian and spinal ganglia there is great capillary dilatation, and the spaces between the cells are filled with a large number of round cells. There is slight proliferation of the capsular endothelium, and the nerve cells appear shrunken in some places, but the changes present can hardly be considered as characteristically those described by Van Gehuchten and Nélis. There are numbers of large pigmented bodies, apparently degenerated cells distributed throughout the ganglia, notably in the Gasserian. Neither the Wolters-Weigert, nor the Marchi methods show the presence of degenerated nerve fibers.

The heart muscle shows beginning fatty degeneration, the liver and spleen are greatly congested, the lung shows congestion and escape of erythrocytes into the alveoli, and the kidney is intensely congested, and shows slight swelling of the epithelium of the tubules.

A review of the findings in this case shows that the lesions agree in general with those which have been previously described in rabies, but while strongly suggestive in a case with so suspicious a history, taken alone without the animal inoculations, they would hardly justify a positive diagnosis.

None of the changes found are characteristic of rabies alone, but each may be present also in other diseases. The lesions of the ganglia considered as specific by Van Gehuchten and Nélis have been found by Crocq in the vagus ganglion of a child dead from diphtheria, by Spiller in a case of endothelioma of the Gasserian ganglion, by Ravenel in the ganglion of a cow with the so-called "forage poison" (epizoötic), and by Burr and McCarthy in a case of neuritis. In a case of acute ascending paralysis, Spiller found general perivascular round cell deposit and small hemorrhages throughout the nervous system, while in the lumbar region of the cord the ganglion cells were surrounded by round cells, and many of them were destroyed, giving a picture much like the rabic tubercle of Babes. Sano, in a case of rabies in a woman fifty-one years old, failed to find the ganglionic changes of Van Gehuchten and Nélis, or the rabic tubercle of Babes. The case here reported was complicated by alcoholism, which would account at least in part for the perivascular round cell deposit, the thickening of the pia-arachnoid, and the pigmentary degeneration of the nerve cells, The examination was made so long after death, however, that it is hardly possible to form a definite opinion as to the cell changes.

From the study of the literature of the subject, together with that of this case, it seems justifiable to conclude that neither the ganglionic changes of Van Gehuchten and Nélis nor the rabic tubercle of Babes is absolutely characteristic of human rabies, though their presence in a suspicious case may be of considerable diagnostic importance. The value of these changes in the nervous system of a dog suspected of rabies is not yet entirely decided, but when found in a case otherwise suspicious they are at least strongly suggestive, and they should invariably be sought for, at any rate until we acquire some more definite information upon the subject.

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# PSYCHOMOTOR HALLUCINATION AND DOUBLE PERSONALITY IN A CASE OF PARANOIA.

By William Pickett, A.M., M.D.,

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Bernard W., a German by birth, was admitted to the Philadelphia Hospital, June 18, 1896. He was then thirty-five years old and had followed the trade of boiler-maker. His family history is unknown. His wife stated that in 1893 Bernard had received a severe blow on the head from a falling log; that early in 1895 he began to complain of pain in his head, heard vague sounds continually, was sleepless, restless, and had fears of harm and misfortune.

He had a number of outbreaks of excitement in which he destroyed furniture at home. He would explain to his wife that these outbreaks were due to "nervousness," and he would advise her to go out of the house lest he do her some injury. After a time his fears took the form of a fixed delusion that certain fellow workmen at Cramps' shipyard, where he had worked, were "robbing him of a patent on a ship" which he had devised, but which he "was too poor to put through."

This delusion was the prominent feature of his case on admission to the hospital; and it has persisted, though now overshadowed by the notions which we shall proceed to explain.

In August, 1898, it was first observed that Bernard was continually uttering in a mechanical way, certain strange expressions—the one which most frequently recurred sounding like "Boon Knecht." When asked what this meant he replied, "I don't know"; why he said it—"I don't say it." Urged to explain, the patient insisted, "I do not say these words, but the man on my back says them." He added that this man on his back does various things with his, the patient's, body, moving his arms, as well as his lips and other organs of speech. Recently Bernard has gone so far as to set aside a portion of his meals, regularly, for the nourishment of this imaginary host on his back.

For a long time this patient's strange belief was regarded merely as one of the "freaks" of persecutory delusion. It was about two years ago, when at the instance of Dr. Dercum, I was looking up the literature of the subject embraced under the title of this paper, that it dawned upon me that the involuntary or "forced" movements of arm or lips in Bernard's case are instances of what certain French writers have called psychomotor hallucination,—the utterance of words ("Boon Knecht," etc.) being *verbal* psychomotor hallucination,—while Bernard's delusion that these weird influences causing him to speak, etc., are due to a man on his back, is what these French writers call "doubling of the personality."

The explanation given by these writers (Séglas, Ballet) is that as ordinary hallucinations are ascribed to excitation of cortical sensory centers, and are called *psychosensory* hallucinations, so these strange motor phenomena, as in Bernard's case, arise from excitation of cortical motor centers, wherefore they may be called psychomotor hallucinations. The angular gyrus being irritated, the patient sees visions; the hinder part of the first temporal convolution, he "hears voices"; the foot of the third frontal convolution, he *feels words spoken silently,* in his head,—or in other parts of his body,—or he may even feel his organs of speech moved, and hear words uttered by his own lips, to the patient's surprise, since he has not consciously conceived or willed these utterances. The silent "interior spoken word" and the unconsciously uttered word arise by the same mechanism; both are psychomotor hallucinations.

Ordinary (sensory) hallucinations are promptly "exteriorized,"—the voices *heard* are ascribed to outside agencies, etc.,—but the psychomotor hallucination impresses the patient as being due to a mysterious agency within himself; and so in time he forms the conception of a new, strange being inhabiting his body or in intimate association with his body. This is the "double personality" of the French, and is exemplified in our patient with "the man on his back."

The term "double personality" is unfortunate, since it has been employed as a synonym for "double consciousness" in the sense of alternating consciousness. We might suggest that some such term as "accessory personality" would be expressive of the condition which we are now considering.

*History of the subject.*—Baillarger (1845) from a study of dreams, divided hallucinations into two groups, (1) those without sound, which he called "psychic"; (2) those with sound—ordinary "voices" of the insane—which he called psychosensorial hallucinations.

Kahlbaum (1866) in classifying hallucinations recognized one kind which he called "abstract" or apperception-hallucination, which is apparently identical with Baillarger's psychic hallucination; and Hagen (1868) had a similar conception in his "pseudo-hallucination."

Tamburini (1881) was the first to apply the modern doctrine of cortical localization to the explanation of hallucinations. In a lecture delivered at the Reggio Asylum, before the class in psychiatry of the University of Modena, he declared that "hallucinations result from an irritative state of the sensory centers of the cortex." Tamburini cited various clinical observations in support of his theory, among them a case of Gowers' in which visual hallucinations in the field of a blind eye were apparently explained, as was the blindness, by the finding of a lesion in the cortical center of vision.

It may be useful in this connection to interpolate a few remarks on what may be called the mechanical theory of hallucinations. It is not new.

Bright, in the *Guy's Hospital Reports* for 1837, mentions a case in which hallucinations of sight followed two apoplectic strokes, and at autopsy a lesion of one corpus geniculatum was found.

Passing by various other instances, we may mention de Schweinitz's case (1891) of left lateral hemianopsia with hallucinations in the dark half-fields. Dercum performed the autopsy in this case, and found gummatous infiltration of the right optic tract.

Spiller has had under his observation for about a year a case of fracture of the skull over the left temporal lobe, in which sensory aphasia and auditory hallucinations are the joint results of injury to the auditory center.

Dr. Spiller has called my attention to a case reported by Sèrieux and Mignot in which cortical deafness, paralexia and hallucinations of hearing were all ascribed to hydatid cysts found in the brain at autopsy.

Regis, in 1881, laid stress upon the significance of unilateral hallucinations, as arguing a unilateral and mechanical origin of them; and he cited numerous instances in which demonstrable lesions of eye, ear, etc., had set up hallucinations. Regis insisted, however, that the intervention of the intelligence is necessary to give form and character to the hallucinations, for we know that they are related closely to the patient's thoughts.

Séglas (1888) in one of his lectures, exhibited a woman who, beside ordinary hallucinations of hearing, recognized an "interior epigastric voice" which said vile things *but without sound.* Séglas also mentioned a patient who felt his lips and tongue compelled to move in speech, sometimes audibly; and another patient who not only spoke, but wrote, words which came from the stomach. He stated that Griesinger long ago referred to the case of a woman who uttered thoughts that she had no intention of saying, and in a voice different from her ordinary tone; and that Baillarger spoke of a patient who moved her lips a little in time with verbal hallucinations.

Séglas concluded that these phenomena belong to Baillarger's psychic hallucinations; that they may be referred to the motor cortex; and that, in distinction from ordinary hallucinations, the proper name for them is psychomoter hallucination.

In 1891 Ballet took up the subject of psychomoter hallucinations in a lecture at Saint-Antoine. He exhibited a Swiss stonecutter who in 1873 had begun to hear voices talking about him, also to hear "interiorly by thought communication," feeling his own thoughts controlled. In the course of six months the patient had conceived the idea that an individual, whom he named Lenoir, was the source of these sensations. In writing, the patient felt his hand deviated by Lenoir; and he constantly communicated with Lenoir, not by voice but, as the patient expressed it, he "felt Lenoir speak."

Ballet identified these phenomena in his patient with the psychic hallucinations of Baillarger and the psychomotor hallucinations of Séglas.

Séglas in 1895 announced his more mature conclusions upon this subject; he had come to believe that the persecutory insane who have pyschomotor hallucinations form a distinct type of paranoia; and that it is only in this "psychomotor variety" of paranoia that double personality arises through the patient's efforts to account for his strange experiences.

Séglas in this paper adopts the theory of Cramer and of Klinke regarding the rôle of the muscular sense in motor hallucinations. These writers had taught that excitation of the centers in which muscular memories are stored causes the inaudible variety of motor hallucinations; while a transference of such muscle-sense hallucinations to the motor centers causes the actual utterance of words, or the movement of other parts. But Séglas still adhered to his original view that the motor cortex is their primary source. The muscular sense centers take cognizance of the involuntary motions excited; and as the muscular sense is an important element of the general physical consciousness (the "cœnesthesia"), the excitation of these centers goes far toward establishing the new personality. As Dercum puts it, the new personality arises by a "hallucination of the cœnesthesia."

*Conclusions.*—The writer's own view of these matters is, that we may accept the theory of psychomotor hallucination by reason of its plausibility; that psychomotor hallucinations are not so rare as we have supposed; that double personality, however, is a very rare sequence of them, and when it is present it is an accidental conception born of ordinary processes of reasoning over strange sensations.

In the case of J. O'N., an intelligent paranoiac committed to the Norristown Hospital by the writer a year ago, both verbal-motor and common-motor hallucinations were present in abundance; but the patient ascribed them to outside agencies. I believe that psychomotor hallucinations are often thus exteriorized. Séglas suggests that the medieval "possession by a demon" (demonomania) belongs partly in the category of these cases, and this seems probable; but if accessory personality arose by an unconscious physiologic process involving the muscular sense, it would be as common now as was the "internal demonopathia" of the old writers; its prevalence would hardly vary at different epochs. It seems more likely that the idea of possession by a demon, being considered rational in the Middle Ages, was resorted to by patients to explain motor, and other sensations which to-day are ascribed by our patients to outside agencies such as hypnotism, electricity, etc.

Nevertheless Séglas' and Ballet's efforts to apply the teachings of modern cerebral physiology to the elucidation of psychiatric problems must be commended by every psychiater; and their ingenious analysis of these cases of psychomotor hallucination with double personality, constitutes one of the fairy tales of science.

# Society Proceedings.

## NEW YORK NEUROLOGICAL SOCIETY.

January 6, 1903.

The President, Dr. Pearce Bailey, in the chair.

*A Marked Case of Exophthalmic Goiter.*—Dr. J. Arthur Booth presented a married woman, twenty-four years of age, exhibiting exophthalmic goiter to a marked degree. The symptoms had appeared four years ago, the right eye being first affected. She presented the rapid pulse and nervous symptoms characteristic of this disease. Dr. Booth purposed having the sympathetic exsected on each side, and would report upon the case later.

Dr. M. Allen Starr said that the tension of the patient's pulse was very high, and the pulse itself was small. This interested him, because in the majority of cases he had seen there had been a decided diminution of arterial tension. This had suggested to him the propriety of using thyroid in cases of high arterial tension, especially in arteriosclerosis and allied conditions. Of late, he had tried this, and had been pleased with the results.

Dr. William M. Leszynsky said that in a recent journal article the statement had been made that the goiter would be practically obliterated for the time being if the patient, while lying down, made an effort to draw the head forward. The pressure of the sternomastoid and other muscles seemed to cause a complete obliteration of the goiter. He had tried this experiment in two cases, with the results stated.

Dr. Booth said that at one time he had been of the opinion that many of these cases presented a high tension pulse, and many of them seemed to be helped by aconite, yet this condition did not seem to prevail in the majority of cases. He had not tried the experiment spoken of by Dr. Leszynsky. He had seen four operations for excision of the sympathetic done, and in two of them the effect on the goiter and on the pulse was prompt and very marked.

Dr. W. B. Noyes said that this operation was certainly a very dangerous one. He had recently had a patient operated upon in this way at St. Luke's Hospital. The operation was rather prolonged, and the patient died on the table without the usual signs of shock.

Dr. Booth said he had been present at that operation, and the case had impressed him as being a very bad one for this operation.

*A Peculiar Affection of the Terminal Phalanges of the Hands.*—This case was presented by Dr. B. Onuf. The affection first began about eleven years ago, some thickening of the terminal phalanx of the middle finger of one hand being first noticed. The corresponding finger of the other hand was next affected, and gradually the disease involved the other phalanges. Subsequently the nails presented transverse ridges, and became very brittle. At the present time, there was very marked thickening of the terminal phalanx of the thumb, and all of the terminal phalanges were in a state of semi-flexion, and showed moderate thickening. There was no specific history, and the examination of the lungs was negative. There was nothing pointing to syringomyelia. Several radiographs of the case were presented. These showed that there was almost complete absorption of the entire terminal phalanx of the thumb and of the middle and index fingers. A radiograph of the foot showed similar changes, though less marked. The disease appeared to Dr. Onuf to be a degenerative one. Radiographs of five phthisical patients having clubbed fingers had been studied by him, but they showed nothing like the same changes as the case just presented.

Dr. Starr said that about seven years ago he had taken photographs of a woman at his clinic who presented a similar condition. The case was not understood at the time, and the photographs were taken because it was considered a curiosity.

*Spastic Paraplegia.*—Dr. George A. Lawrence presented specimens from a case of spastic paraplegia that he had under observation since November 9, 1895. The patient gave a history of scarlet fever and of dorsal Pott's disease in early childhood. He was able to attend to business until the summer of 1895, when he fell downstairs and became unconscious. This was followed by severe epileptiform seizures. Examination showed no paresthesia or anesthesia, but there were marked ankle-clonus and fibrillary twitchings of the muscles in the legs, with jerking of the limbs at night. The man received iodide of potassium in doses as high as 200 grains, three times a day, and also strychnine sulphate in doses varying from 1-100 to 1-50 of a grain. He improved greatly under this treatment, and by the fall of 1896 was going to business. In the following March there was another exacerbation. In September, 1898, he was able to return to business. On March 15, 1899, there was considerable pain in the chest and a drawing up of the limbs was still more marked. In October of that year there were marked fibrillary twitchings of the muscles of the legs and thighs. In December, 1900, there was another exacerbation. On October 2, 1902, the upper extremities were becoming involved, and the patient was growing emotional. On December 1 the heart action and breathing were rapid, and he was exceedingly nervous. On December 26, 1902, death occurred. A post-mortem examination of the brain and spinal cord was alone permitted. This was made by Dr. Strong and Dr. Lawrence the following morning. The spinal canal was found to have been encroached upon at the site of the old kyphosis, and there was much thickening of the anterior surface of the dura mater. The anatomical diagnosis was spastic paraplegia due to a chronic myelitis. The microscopical examination had not yet been made.

*Address of the Retiring President.*—Dr. Joseph Collins, in retiring from the chair, reviewed the scientific work of the society during the two years he had been its president.

*Inaugural Address of the President.*—Dr. Pearce Bailey selected as the theme for his address, "Fracture at the Base of the Skull." He said that from the uniformity in localizing signs the conclusion was that the brain lesion accompanying this fracture must also be quite constant. Neurological examinations were often made with difficulty, because the patient was comatose. The cranial nerve injuries were usually multiple and came on immediately, or within a few hours. When appearing still later they were difficult of explanation. Paralysis of the olfactory nerve was sometimes the only focal symptom, but it was usually absent altogether. Blindness and amblyopia in fractures of the base were usually due to fracture extending into the optic canal. It was probable that amblyopia might also be caused by hemorrhages at the base of the skull, which push their way into the optic canal and compress the nerve. Visual disturbances following fractures were always alarming symptoms. Traumatic affections of the optic nerve were frequently accompanied by oculomotor palsies. The frequency with which the third, sixth and fourth nerves were involved was in the order named. The sixth nerve and the seventh were often injured, and this was commonly associated with deafness. It was rare for oculomotor palsies to persist longer than a few months. In most of the cases of traumatic ptosis that he had seen there had usually been sufficient weakness of the affected eyelid to enable one to determine which lid had been involved. Generally the dilated pupil corresponded with the side having the greater brain injury. Of five cases presenting equally dilated pupils only one recovered. The facial was the most frequently involved single nerve. Paralysis of the extremities showed itself more commonly as a weakness or as a hemiplegia. Paraplegia proved almost invariably fatal. The knee-jerks were frequently diminished or lost at first, but later returned, and might even become over-active. Convulsions were very rare in fractures at the base, From the initial profound coma a large number of persons who die never emerge, but profound and persistent coma was rare in cases which recover. The most characteristic mental state in these cases was semicoma. A fatal result was to be looked for in more than one-half of the cases of fracture at the base. A collection of 494 cases showed a mortality of 57 per cent. In probably 95 per cent of the fatal cases death occurred within five days of the receipt of the injury. Age did not seem to be an important feature in prognosis. Alcoholism affected profoundly both the immediate and ultimate prognosis. With regard to the ultimate prognosis, the speaker said that the literature on this subject was meagre. He had been able to obtain the ultimate result in 14 out of his 28 cases. In all of the cases there was a period of at least one year after the injury. In general, it might be said that in the majority of cases the patient would be as well as before the injury at the end of the first year. In a certain small proportion of cases, however, there were certain changes in disposition or mentality. Irritability of temper, quarrelsomeness, headache and diminished resistance to alcohol were noted in some of those suffering from diseases, such as Bright's disease, which affect the arteries. Epilepsy did not seem to be a consequence of fracture at the base of the skull. If brain injury figured in the etiology of insanity, such patients should be found in insane asylums. It was probable that there were 500 cases of fracture at the base occurring annually in New York city, and that in 150 of these cases recovery occurred; hence, if such an injury were a common cause of insanity these persons would be found in the insane hospitals of this vicinity. Dr. A. E. Macdonald had kindly investigated this question for Dr. Bailey at the Manhattan State Hospital, but had been unable to find more than one such patient in a thousand. The conclusion seemed to be that fracture at the base was a negligible quantity in the etiology of insanity.

*The Daily Rhythm of Epilepsy and its Interpretation.*—Dr. L. Pierce Clark read this paper. He said that in 1897 he had collected and tabulated 9,545 eqileptic seizures by hours. The greatest number of seizures occurred at 4 a. m. Since then he had collected cases occurring in both sexes at the Craig Colony, and had tabulated 150,000 epileptic seizures. During the greater part of this study sedative treatment was at a minimum. The great majority of the patients were in bed, in the first group, and slept from 7.30 p. m to 5.30 a. m. There were three fairly marked curves; that of early night, or 9 p. m.; that of early morning, or 4 a. m., and that of noon. Another class studied was among the out-door workers at the colony. The three main curves were much the same as in the other group, and, as in that group, there were fairly marked secondary curves at 9 a. m. and 5 p. m. In another chart, comprising the whole of the 150,000 seizures, the secondary curves were still more apparent. It was found that the seizures were about equally divided between the day and night. Sleep was deepest during the first hour, then became lighter for an indefinite period, and then deepened again and lightened once more about one hour before awakening. In certain cases in which deep and perfect sleep did not come at once various crises occurred. During the deepest sleep of the epileptic he had never been able to elicit the knee reflexes without adopting sufficiently harsh measures to awaken the person. The bromides diminish the afferent impulses to the cerebral cortex, and are aids to cerebral inhibition. At noon the epileptic became fatigued and was in a condition comparable to light sleep. Moreover, at this time the heaviest meal was taken, and auto-intoxication was at the maximum. Epileptics should not be idle; they should have a simple but not a monotonous occupation. This study showed that the twenty-four hours were roughly divided into eight-hour periods.

Dr. B. Sachs asked for further information regarding rhythm in the individual epileptic; also if one of the reasons the attacks were so apt to occur in the early morning hours, was that the air of the sleeping apartment was especially vitiated at this time. He would also like to know if it were safe for epileptics to engage in occupations which would place them in danger should a seizure occur.

Dr. M. Allen Starr said that he had noticed that in the individual there was a tendency for the seizure to return at a certain hour. He had just seen a patient who for sixteen years had never had the attack except between six and seven o'clock in the morning, but who for the first time yesterday had had an attack in the afternoon. This fact seemed to him of some importance in the treatment. Attacks occurring in the early morning hours seemed to point to a weakened circulation in the brain, and hence in such cases he was disposed to combine such remedies as digitalis and strophanthus with the bromides. In some of these cases he thought benefit was derived from awakening the patient early in the morning and giving some hot stimulant.

Dr. W. M. Leszynsky said that in cases of nocturnal epilepsy the rhythm had been considerably changed by the bromide treatment. The attacks would often disappear at night and occur in the daytime, when they had not been so before this treatment was instituted. Mention was made of a man whose attacks had always been at night, while taking bromide at night, and whose attacks first appeared in the daytime when the bromides were given during the day.

Dr. J. Arthur Booth said that he, too, had observed this change in the rhythm as a result of treatment.

Dr. I. Abrahamson asked if Dr. Clark had made any investigations on the metabolism and blood pressure at the time of maximum seizures.

Dr. Smith Ely Jelliffe said that recent investigations on blood pressure had shown that it was at a minimum in the early morning hours. It had been claimed that there was more or less rhythm in the leucocyte count at the different periods of the twenty-four hours.

Dr. Clark said that there was no doubt that investigation of the metabolism and blood pressure would show them to go hand in hand with that condition expressed only too indefinitely by the term "auto-intoxication." Alterations in blood pressure, he thought, varied with the depth of sleep. The rhythm of the epileptic seizures were certainly greatly influenced by treatment, as well as by diet, exercise and other things. He had known many epileptics to carry on the trade of carpenter for a long time without meeting with any accident, but the latter was prone to come when they became so accustomed to their work as to relax their guard.

## PHILADELPHIA NEUROLOGICAL SOCIETY.

### January 27, 1903.

The President, Dr. John K. Mitchell, in the chair.

*Some New Reflexes.*—Dr. D. J. McCarthy demonstrated a new spino-muscular phenomenon. He had found that tapping over the lower lumbar enlargement of the spinal cord, when the first, second or third lumbar vertebra is reached there is a simultaneous contraction of the semitendinosus and semimembranosus muscles.

He also demonstrated that in the case of muscles having a tendonous origin and insertion a reflex may be elicited by striking at either end. This was shown in the adductor of the leg and in the sartorius.

Certain reflexes about the ear and jaw were also referred to.

*Paradoxical Reaction of the Pupil in Accommodation.*—Dr. William G. Spiller referred to the observations of Vysin of what he had termed perverse reaction of the pupil in accommodation. That observer had seen the phenomenon in two cases and probably no other cases are to be found in the literature. The reaction consists in contraction of the pupil on fixing a far object and in dilatation on fixing a near object. One of the cases reported by Vysin was a traumatic neurosis and the other a case of migraine. In the second case the perverse reaction was present only during the attack.

Dr. Spiller said he had seen this reaction in three cases. One case was that of a woman with organic disease of the spinal cord. The second case was one of mild hysteria, and in this and the following case the observation was confirmed by Dr. W. C. Posey. The third case was in a perfectly healthy person. Dr. Spiller had found that in at least two of these cases, when the patient with one eye covered looked at the point of the nose with the other eye the pupil dilated, while if both eyes were open there was the ordinary contraction of the pupil in convergence. The paradoxical reaction is not the contraction of the pupil which occurs when the eyeball turns upward to which Dr. McCarthy has called attention. This may be an element in the so-called Piltz-Westphal reaction, which consists in contraction of the pupil in forcible closure of the lids, as when the lids are closed the eyeball turns upward.

*Two Cases of Huntington's Chorea with the Pathologic Examination in One Case.*—This report was made by Drs. C. W. Burr and D. J. McCarthy. The first patient was seen in 1889. The father had had marked Huntington's chorea and was quite demented. He had four sons. The first died before the age of eighteen years of acute infection. The second son is fifty-two years old and has had Huntington's chorea since 1888, but mental symptoms did not appear until two or three years ago. The third brother developed Huntington's chorea ten years ago and committed suicide. The fourth brother, now forty years of age, is in the early stages of the same disease.

Another clinical observation made was that during the fall of last year the patient developed symptoms of acute excitation with delusions of grandeur and marked motor weakness. This continued two or three months, and then practically disappeared, and the patient is able to be about with some minor delusions.

The man from whom the specimens were obtained was aged forty-two years. He had been an inmate of the Insane Department of the Philadelphia Hospital. A sister aged forty-eight years has had chorea for five or six years. Other members of the family had suffered from the same disease. Dr. McCarthy described the pathological changes he had found in the brain from this case.

Dr. William G. Spiller said that some years ago he had examined the specimens from a typical case of Huntington's chorea in the service of Dr. Dercum, and had found the lesions very slight. He doubted very much whether the lesions found could be considered as the direct cause of the chorea.

*Hemiplegia with Sweating on the Palsied Side of the Head and Opposite Shoulder.*—This case was exhibited by Dr. Wilfred W. Hawke. The patient was a white male aged fifty-two years. He had had a stroke affecting the right side in 1885, and later had a second stroke affecting the left side. The sweating involved the right side of the face and the left shoulder.

*A Case of Multiple Fibromata Confined to the Internal Plantar Nerve.*—This paper was read by Drs. Wm. J. Taylor and William G. Spiller. (See page 204.)

*The Histopathological Changes in the Cerebral Cortex of Epileptics and their Interpretation, with Demonstration of the Lesion.*—This paper was read by Drs. L. Pierce Clark and T. P. Prout.

Dr. William G. Spiller said that he had understood from the paper that the changes found were in the granular layer and not in the pyramidal cells or in the cells of Betz. From Dr. Prout's remarks he gathered that the action of the bromides was supposed to be on the the sensory portion of the reflex arc, and that therefore the cells of the granular layer, according to Dr. Prout, might be regarded as sensory.

He asked whether the changes found were more distinct in any one portion of the cortex; for instance, in psychical epilepsy were the cells of the frontal lobes more markedly affected; or in epilepsy with a visual aura were the cells of the visual cortex more altered? He called attention to the fact that the bromides had been found to produce changes in the nerve cells of the cortex, but he was not aware that these changes were especially marked in the granular layer.

Dr. William Pickett referred to the statement made, that the gravity of epilepsy should be measured by the mental symptoms. Trousseau has been credited with originating the teaching that the mental symptoms are more prominent in minor epilepsy than in the major form. Esquirol also pointed out that epileptic insanity progresses more rapidly in cases with the minor attacks than it does where major attacks occur early. Dr. Pickett had himself seen a number of cases of epileptic insanity associated with minor attacks.

Dr. T. P. Prout said with regard to the distribution of the lesions in epilepsy, that his studies had shown that the lesions were not limited to any one portion of the cortex but were general in their distribution. The special involvement of certain areas may in the future be proven, but nevertheless the whole cortex is involved.

With regard to the second, or granular layer, while he hesitated to ascribe distinct sensory functions to this layer, yet he felt that he was justified in considering that the cells of the granular layer have a sensory character in the main. In the experiments made by Prus some three years ago, this experimenter found that after penciling the surface of the motor cortex with cocaine he was no longer able to produce epileptic explosions by cortical irritation with the electric current. He was still able, however, to get a motor response in the corresponding muscles in the extremities. One of his conclusions was that epilepsy is essentially a sensory phenomenon.

Dr. L. Pierce Clark said it was unfortunate in many respects, especially as to accuracy in measuring the severity of the epilepsy, that the character of muscular movements in the fit was made the basis of classification. Many types of seizures in which little or no appreciable convulsions occur are accompanied by psychosensory changes of the most regrettable sort. Indeed Esquirol and Delasiauve had long ago declared the so-called minor attacks most disastrous to mental integrity. Nervous discharges in the re-represented frontal brain centers can be attended and followed by mental changes only of the tissues undergoing degradation, which are impairment in judgment, self-control and memory. To illustrate the modern trend of emphasizing the sensory and psychosensory changes in epilepsy, at the Craig Colony it had been a matter of custom for some time to base the severity of prognosis of the individual case upon the mental changes present, rather than upon the type of convulsive movements.

## CHICAGO NEUROLOGICAL SOCIETY.

January 22, 1903.

The Vice-President, Dr. H. H. Donaldson, in the chair.

*Structure of Neuroglia.*—Dr. G. Carl Huber, of the University of Michigan, reported observations of his, extending over a series of years, embracing a study of neuroglia tissue of different classes of vertebrates, made especially with the Benda's sulpha-lizarate toluidin blue method. The research was undertaken with a view of harmonizing the various views expressed concerning the structure of neuroglia tissue and of obtaining data which must of necessity be at hand in order that experimental work involving structural changes in the neuroglia tissue might be undertaken. The report embraced observations made on the neuroglia tissue of man, dog, cat and rabbit among the mammals; dove, tortoise and frog, in all of which the neuroglia tissue of the white matter of the spinal cord was especially considered, since it was found that the relation of the structural elements of this tissue might here be more clearly made out, and its relation to other structures more exactly defined. The results obtained were controlled by observations on the gray matter of the spinal cord and brain.

The following general conclusions were reached: The neuroglia tissue of all vertebrates consists of neuroglia cells and neuroglia fibers. The cellular elements vary greatly in size and shape, depending in the main on the spaces which they occupy. In the majority of the neuroglia cells a granular cytoplasm can be clearly made out. This varies greatly in amount, at times forming only a narrow zone around the nucleus; again present in much larger quantity and presenting very distinct protoplasmic branches. In certain cells, which are designated as "free nuclei," no distinct cytoplasmic covering can be seen. The nuclei of neuroglia cells vary in size, shape and structure. The majority of them may be spoken of as vesicular nuclei with chromatin in the form of a number of relatively large granules, variously disposed. Other nuclei, usually the smaller and smallest seen, present the appearance of compact masses of chromatin, taking the stain relatively deeply. Transition forms between the two varieties of nuclei specially mentioned may readily be made out. The neuroglia fibers show very definite relation to the neuroglia cells, being imbedded in the peripheral layer of their protoplasm. With the method used, whenever the preparation is properly differentiated, the neuroglia fibers could be traced through the protoplasm of neuroglia cells without interruption. In imperfectly differentiated preparations, they often appeared as processes. The small, deeply-stained "free nuclei" and the free nuclei showing a vesicular structure, show no definite relation to neuroglia fibers, and are looked upon as undifferentiated and undeveloped neuroglia cells.

Microscopic preparations substantiating the statements made were demonstrated.

*The Mesoblastic Origin of the Neuroglia Tissue.*—Dr. Shinkishi Hatai, from the Neurological Laboratory of the University of Chicago, reported upon the origin of this tissue. He said: On examining the cross sections of the central nervous system of the white rat and mouse, two kinds of nuclei, differently characterized, are easily recognized. One kind coincides in structure with that found in the nerve cells, while the other resembles very much in size, shape and staining character, and in an internal structure, the nuclei found in the walls of the capillaries. These nuclei, so called, are always surrounded by a small amount of cytoplasm. These facts led the writer to a systematic study of these structures in order to determine their origin within the central nervous system.

For the investigation new-born white rats and mice were used. The tissues were fixed in Carnoy's mixture, and the paraffin sections were made and stained with toluidin blue and erythrosin.

One group of the nuclei, which resembles in structure as well as in shape those of the nerve cells, can be identified without difficulty as derived from the ectoblast. For convenience, Dr. Hatai called these nuclei Type I. The identification of the other group of the nuclei, or Type II, is, however, difficult, since these nuclei have no similarity to the nuclei of the ectodermal derivatives. On examining the capillary wall, he noticed very frequently a nucleus projecting outwards, and in some cases these nuclei were isolated from the capillary wall but lay close to it. Between these two appearances various intermediate stages could be easily found. In some cases a tip of the nucleus is still attached to the capillary wall while the rest of it has become separated. This appearance shows very plainly the migration of the nucleus from the capillary wall into the surrounding tissue. There is no danger of confusing these nuclei with those of the leucocytes which escape from the capillary wall and exhibit ameboid movements, since they differ from them both in structure and size: the former being very must larger than the latter. As soon as the nuclei are separated from the capillary wall they migrate away from the capillary.

The following observation is in favor of the above conclusion. A large number of the mitotic figures in various phases are often visible in the nuclei of the capillary wall. In such a locality where the mitoses are abundant the nuclei are so closely placed that in some cases the one nucleus overlaps the others, the outermost nucleus projecting outwardly, thus showing the first step of the migration.

From the above Dr. Hatai thinks there is no doubt that the nuclei which he has called Type II have been derived from the capillary wall and migrated away from it. The identification of these nuclei which have migrated from the capillary wall with neuroglia nuclei is difficult. Since it has been shown that a small amount of connective tissue is present in the central nervous system in both young and old animals, this connective tissue might have been formed by the nuclei described, but there is no evidence to support the view that this could occur. The large number of nuclei which have migrated from the capillary walls suggests at once that some of the neuroglia tissue is supplied by these nuclei. This idea is favored by the fact that similar neuroglia nuclei to those migrated from the capillary wall or mesodermal in type, exist in the adult animal.

According to Kühne and Chittenden, the neuroglia fiber is produced by the chemical transformation of cytoplasm into neurokeratin. Chavalier further showed that the same chemical substance exists also in the medullary sheath of the peripheral system. The presence of the neurokeratin meshwork in the peripheral nerve has been shown by histologists also. If it is true that the entire medullary sheath in the peripheral system is formed exclusively by the mesoblastic cells, one can assume that there is here a chemical transformation of mesoblastic cytoplasm into structures characteristic of ectodermal cells. This evidence favors Dr. Hatai's hypothesis that the nuclei which have migrated from the capillary wall might be transformed into the neuroglia tissue and give rise to neurokeratin. Since we have no evidence that Weigert and Benda's technic brings out the neuroglia fibers of ectodermal origin only, these methods can hardly touch the point mentioned above. Dr. Hatai therefore concludes at the present moment that the nuclei which migrate from the capillary wall into the central nervous system are one source of the neuroglia tissue. The reasons for this conclusion may be summarized as follows:

(1) The great number of these nuclei; (2) neurokeratin may be formed not only by the cytoplasm of the ectodermal cells, but also by the cytoplasm in mesodermal cells; (3) presence of the nuclei of mesodermal type in the central nervous system of the adult animal; (4) ordinary connective tissue nuclei present in the central nervous system are there in much smaller numbers than the nuclei which have migrated from the capillary walls; (5) there is no evidence that the neuroglia fibers stained by the modern neuroglia technic are of ectodermal origin only; (6) since the cytoplasm undergoes very easily morphological and structural alterations while the nucleus is more resistant, it follows that the nuclei are the better indication of the genetic relationship of the cells; (7) Capobianco and Fragnito, who observed the nuclei which migrate from the meninges into the central nervous system and believe these nuclei form a part of the neuroglia tissue, were unable to trace the successive transformations of the former to the latter. This observation of Capobianco was made on the spinal cord of *Mus decumanus.*

# Periscope.

### MONATSSCHRIFT FUR PSYCHIATRIE UND NEUROLOGIE

(Bd. 13, 1903, Hft. 1, Jan.)

1. The Origin and Course of the Oculomotor Nerve in the Mid-brain. N. MAJANO.
2. Hysterical Attacks Occurring Late in the Course of Epilepsy. F. S. HERMANN.
3. The Forensic Significance of Normal and Pathological Drunkenness. A. CRAMER.
4. Scopolaminum (Hyoscinum) Hydrobromicum. BUMKE.

1. *Oculomotor Nuclei.*—Majano has made a careful study of the oculomotor nuclei in the brain of a man dying at the age of forty-eight years, who presented during life the following conditions. Between eighteen and twenty years of age he had had paralysis of the left eye with slight ptosis. About eighteen months before death he developed symptoms of psychosis. He had numerous stigmata of degeneration; there was slight paresis of the right external rectus, and the left eye only moved imperfectly inward and upward or downward. The ability to lift the left upper lid was also impaired. The right pupil was narrow, the left greatly dilated. The light reflex was sluggish on the right and lost on the left. There seemed to be slight paresis of the facial and hypoglossal nerves. There was also ataxia, Romberg's symptom, diminished knee-jerks and delusions of grandeur. The patient rapidly grew worse and died. At the autopsy the left oculomotor and right abducens were noticeably thin. Majano gives a careful description of the histological changes found in the oculomotor nuclei. (1) Darkschewitch's, Edinger's and Westphal's nuclei were normal on both sides; (2) the anterior median nuclei were very slightly affected, the right side more than the left, although both were doubtful; (3) the dorsal portion of the principal nucleus was degenerated in its whole extent with the exception of the left side at the level of the oculomotor decussation. The ventral portion was degenerated on both sides in the region of the decussation. The accessory nucleus of Bechterew was degenerated on the left, and there was a general reduction of the left principal nucleus; (4) the median nucleus of Bechterew showed changes chiefly in the proximal region on the left side. In the distal portions there was no pronounced difference. (5) Bolliger's nucleus in the neighborhood of the trochlear nucleus was unaffected on either side. Majano believes that a careful comparison of these lesions with the clinical symptoms indicates that there is a partial decussation of the oculomotor nerve. He explains the mechanism of conjugate movements of the eye by supposing that the impulse passes from the abducens nucleus to the principal nucleus on the same side at the point from which the decussated fibers arise, which pass into the oculomotor nerve of the opposite side. He discusses carefully the opinions of different investigators, and believes that there can be no doubt that if we deny the existence of decussated fibers in the proximinal portions of the oculomotor, the existence of commissural fibers uniting the oculomotor nerve nucleus must be accepted. (The paper is still unfinished.)

2. *Hysteria and Epilepsy.*—A man, thirty-five years of age, with a

family history of misuse of alcohol, developed epilepsy at the age of fifteen years after an injury. The epileptic attacks were typical. On one occasion, however, he awoke feeling badly, with contractures in all the limbs without pain. These spasms could be overcome by passive movements. There was a motor aphasia without loss of understanding of what was said. Examination of sensation showed entire loss in the lower extremities, with loss of the pain sense in symmetrical parts of the body. The symptoms were dissipated by faradic electricity. Subsequently the patient had a second hysterical attack with somewhat similar symptoms. The second case, a man of thirty-four, with a family history of misuse of alcohol, had had epilepsy since the age of twelve years. This patient developed nervous hysterical phenomena, such as catalepsy, motor aphasia, loss of pain sense and of taste, rhythmic movements of the head, and these attacks lasted throughout the day. Both cases had had epilepsy for a long time; in both the hysterical manifestations were characteristic and differed from the ordinary epileptic attack. It is possible that the previous alcoholic indulgence of these patients predisposed to hysteria.

3. *Drunkenness.*—Cramer discusses the significance of intoxication, and calls attention to the fact that in the German law it is not an extenuating factor of crime. He speaks of the conditions that give rise to it, either congenital, including the degenerates, and the acquired, which includes chiefly neurasthenics. Intolerance to alcohol may be transient, and therefore even if the patient withstands an experimental indulgence it does not prove that during the criminal act he was in a normal condition. He calls attention to various other factors that influence the resistance to alcohol, and then gives the following points for the diagnosis of pathological intoxication. First, the recognition of a more or less pronounced general pathological foundation. Second, the recognition of particularly injurious factors. Third, the pathological manifestations during the period of drunkenness—anxiety, delirium, hallucinations, peculiar motor reactions, the relation of the pupils, terminal sleep, etc., and then reaches the following general conclusions: (1) Intoxication can only be regarded as pathological before the law when it is influenced by certain factors of disease; (2) The physician should only offer expert testimony when these pathological factors are present; (3) Intolerance of alcohol does not indicate that the intoxication is abnormal; (4) In psychopathic individuals habitual drunkenness may cause repeated crimes of the same character; (5) Experimental intoxication gives positive results only when there is habitual intolerance of alcohol, or when peculiar accompanying pathological conditions are produced; (6) The recognition of a disturbance of consciousness is not sufficient to indicate pathological intoxication; the other morbid factors must be recognized; (7) Amnesia or conditions that occur during the attack may be complete or partial; (8) The crimes are usually those of violence; (9) The attack ceases usually with collapse followed usually by terminal sleep; (10) A sluggish pupillary reaction is of importance when present, but its absence does not exclude pathological conditions.

4. *Hyoscine Hydrobromate.*—Bumke has employed hydrobromate of hyoscine as a hypnotic. He used it only subcutaneously in doses of .0005 to .001. Usually it was given at 8 o'clock in the evening; the effects were prompt and satisfactory in all classes of patients, including melancholics. (The paper is still unfinished.)

J. Sailer (Philadelphia).

(Bd. 13, 1903, Hft. 2, February.)

1. Contribution to the Clinical Significance and Pathogenesis of Babinski's Reflex. W. Specht.
2. Contributions to the Comparative Pathological Anatomy of Acute Encephalitis. H. Dexler.

3. Disease of the Posterior Roots with Loss of the Patellar Tendon Reflexes in Tumors of the Brain. W. ERBSLÖH.
4. Scopolaminum (Hyoscinum) Hydrobromicum. BUMKE.
5. The Origin and Course of the Oculomotor Nerve in the Mid-brain. N. MAJANO.

1. *Babinski's Reflex.*— Specht has made careful clinical and literary studies of the Babinski reflex. Clinically he investigated 430 cases, 17 of which had distinct lesions of the pyramidal tracts; 22, doubtful lesions of the pyramidal tracts; 30 were children in ages from one to fourteen days. There was one case of epileptic coma, and one of hysterical paralysis of the legs. The other cases were normal. In the 17 certain cases the reflex was prompt and characteristic. In a few it was possible to obtain the extension of the great toe by striking the back of the foot. In the 22 doubtful cases it was present five times in four cases of Liten's disease, and in a case of flaccid degenerative paralysis of the right leg. In the left leg there were also slight spastic symptoms, and the reflex was present. In the other 17 cases there were such conditions as hydrocephalus, multiple sclerosis, syringomvelia, cerebral tumor, infantile cerebral diplegia; in all the reflex was present. In the case of cerebral tumor there was exaggeration of the patellar and Achilles tendon reflexes, but microscopical examination of the cord showed that the pyramidal tracts were intact. In the 30 sucklings the reflex was obtained in 28 cases. In the other two cases, both children eighteen days old, it was doubtful. In a case of epileptic coma which was repeatedly observed, the skin and tendon reflexes disappeared, and the Babinski reflex appeared during the acme of the condition; it then disappeared and the other reflexes returned. In a case of hysterical paralysis it was absent, and also in 359 cases in which there was every reason to believe the central nervous system was normal. He therefore concludes that the great-toe phenomenon may be regarded as a trustworthy symptom of degeneration of the pyramidal tracts, or in those cases in which it is present and no organic disease exists, it indicates a functional disturbance of the pyramidal tract. He gives a theoretical explanation of the nature of this reflex, which is based upon the fact that in certain disease conditions of the pyramidal tract the sensory irritation is sufficient merely to produce retraction of the toe without retraction of the leg.

2. *Acute Encephalitis.*—Dexler describes some cases of encephalitis that occurred in animals, with the clinical symptoms and pathological changes. Bacteria that bear any etiological relation to the condition apparently were not present. (The paper is still unfinished.)

3. *Tumor of Brain and Tendon Reflexes.*—Erbslöh reports two cases of brain tumor in which the knee-jerks were absent. The first, a man of forty-eight years, had a tumor in the left temporal sphenoidal lobe, which proved to be an angiosarcoma. The pathological changes were found in the extra and medullary portions of the posterior roots, and some degenerated fibers were also found in the posterior columns. The second case, a man of eighty-seven years, showed the same conditions. Erbslöh admits that he is unable to explain why the posterior roots become degenerated in brain tumor.

4. *Hyoscine Hydrobromate.*—The effect of hyoscine upon healthy individuals appears to vary. It usually produces fatigue, profound sleepiness, and occasionally some dryness of the throat. More rarely, however, patients have hallucinations. The repeated employment of it appears to have no cumulative or other injurious effect upon the patient. He believes that it is the best hypnotic and sedative for excited mental conditions. Symptoms of poisoning only occur when high doses are employed (more than 2 Kilo), or in very rare cases of individual idiosyncrasy. It is not known to produce death, and is without influence upon the course and duration of the psychosis.

5. *Oculomotor Nerves.*—Majano continues his article upon the origin of the oculomotor nerve in the mid-brain. He has had an opportunity of examining a number of additional preparations, including a series from a case of tabes who had developed a left-sided ptosis and a paresis of the right rectus. He found that the predorsal tract was impaired in its entire extent on both sides. The principal nuclei were also affected, particularly the dorsal group on both sides. Some of the other nuclei were also involved from a case of microcephalu in which the movements of the eyes and the reactions of the pupils appeared to be normal; also in a case which was not examined clinically, which presented an enlargement of the right optic thalamus and dilatation of the ventricle. For comparative purposese he examined the brain of a *Macacus* in which bilateral destruction of the occipital and a portion of the parietal lobes had been produced. He gives in detail the histological changes found in these preparations, and draws the following conclusions: First, that the sublongitudinal or dorsal tract arises chiefly from the nucleus of the anterior corpora quadrigemina. Second, that the ventricular portion of Meynert's commissure arises from the dorsomedian nucleus of the dorsal corpora quadrigemina. It appears likely, moreover, that the sublongitudinal tract, after arising from the lateral nucleus of the anterior corpora quadrigemina, passes obliquely downward in the region of Meynert's fibers toward the median line, and lies close to the capsule of the nucleus rubra. From this point some of its fibers pass downward and unite with the root fibers of the oculomotor nerve of the same side. The greater part decussate with the fibers of the opposite side, passing obliquely backward and outward and finally uniting with the root fibers of the oculomotor nerve of the opposite side. J. Sailer (Philadelphia).

## BRAIN

(Vol. 25, 1902, No. 3, Autumn.)

1. Degeneration following Lesions of the Retina in Monkeys. J. Herbert Parsons.
2. Note on the Arterial Supply of the Brain in Anthropoid Apes. A. S. F. Grünbaum and C. S. Sherrington.
3. Experiments on the Conductivity of the Spinal Cord rendered Anemic by Compression of the Aorta. M. Loewenthal.
4. Further Observations on a Case of Convulsions (Trunk Fit or Lowest Level Fit). J. Hughlings Jackson and Stanley Barnes.
5. Hereditary Aphasia: A Family Disease of the Central Nervous System, due possibly to Congenital Syphilis. W. G. Stone and J. J. Douglas.
6. Case of External Spinal Pachymeningitis, implicating the Entire Ventral Surface of the Spinal Dura. C. K. Mills and W. G. Spiller.
7. On Changes in the Central Nervous System in the Neuritic Disorders of Chronic Alcoholism. S. J. Cole.
8. Case showing (1) Defective Development of the Dura Mater; (2) Old Meningitis, and (3) Internal Hydrocephalus. Harvey Baird.
9. Pain. James Mackenzie.

1. *Degeneration Following Lesions of Retina in Monkeys.*—The anatomical evidence that the ganglion cells of the retina are comparable to those of the posterior spinal root has but recently been established. The centripetal afferent fibers may, therefore, be rendered easily an object of degeneration experiments by the Marchi method. The work of Pick, Falchi and Baquis in destroying circumscribed areas of retina and studying after-lesions are well-known both to neurologists and ophthalmologists. The results of their studies regarding the relationship of the retina to that of the optic nerve and chiasm and tracts have recently been confirmed by Parsons in experimental studies upon degeneration following lesions of the retina in monkeys. He also suggests explanations of the presence of degeneration in the retina and optic nerve of the sound side; namely that the fiber degeneration

is of collaterals from the injured side, inasmuch as the Marchi shows much smaller and more diffuse degeneration dots. This also held true for the few scattered and much smaller spots on the contralateral tract from injury of the distinctly temporal half of the retina. An arrangement of this kind suggests a possible subservance of some of the hitherto unexplained phenomena of binocular vision in a modified type. However tempting the data for explanation of this sort may be, the evidence of some half dozen monkeys is altogether insufficient for conclusions, and similar research should be pursued further, pathologically and experimentally, particularly as the difference between the human and animal eyes is well known. The author ascribes the degeneration in the third and fourth nerves, that have been found in all these experimental investigations, to artefacts such as Marchi himself found in his early experiments. The rather brief but suggestive experimental work of Parsons should be undertaken upon a more thorough and exhaustive scale in the future; it is a field of practical value, which he rightly claims has been too much neglected in the past.

2. *Arterial Supply in Anthropoid Apes.*—The authors have investigated six chimpanzees and one orang-outang, the former of the species *Troglodytes niger*, from which they conclude that, in the first place, in the chimpanzee and orang an arterial circle of Willis of the human type is met with; although, as shown by Bolk's two orang brains and by one of their chimpanzees, it is not met with invariably. In the second place, they show that in collateral supply of the brain by anastomotic arterial provision, there is, just as with the morphology of the arterial circle, considerable range of individual variation. In some chimpanzees the arterial anastomosis is found experimentally to have sufficient freedom for an occlusion of both carotids maintained for eight minutes, to make no obvious alteration in the faradic excitability of the cortex, at least, in its motor region. In other individuals such occlusion is found to rapidly paralyze the cortex, at any rate, in its motor region, when tested by electrical stimulation. This conclusion harmonizes strikingly with the conclusion drawn by L. Hill regarding the efficiency of the circle of Willis in man. Hill states that sudden compression of the common carotid artery in some men produces epileptic spasm, and ligation of this artery has been followed in some by more or less temporary paralysis on the opposite side of the body; in others the effect is *nil.*

3. *Experiments on Conductions of the Spinal Cord Rendered Anemic by Compression of the Aorta.*—Although definite knowledge of the anatomical integrity of spinal tracts is nearing completion, that of their function leaves much to be desired. The completion of these data however, is necessary for early diagnosis of cord affections in the future. This fact is especially true in the differential diagnosis of all forms of compression paraplegia. The excitability of the gray and white matter of the spinal cord has been the subject of study for a great many years, but the conductivity of the cord to impulses coming from the outside has not been under investigation until Max Loewenthal took up the subject. He has recently given careful study to experimental work of this sort by compressing the thoracic aorta in cats He finds that for an at present unexplained reason "the first effect of arrest of the circulation on the functions of the spinal cord is an increase in the readiness and strength of interneural transmission of nerve energy which lasts about thirty seconds." In other words there exists a hyperexcitability of the interneural segment, and this accounts for the increased conductivity by cortical excitation and not as formerly supposed, by the white fibers of the cord conducting through the anemic area. It is fairly well known that in man, and particularly in animals, motor impulses may pass in a large measure over the so-called extra-pyramidal tracts or Monakow's fasciculus. An impulse traversing this tract after section of the pyramidal fasciculi would, however, necessitate its traveling over sections of gray matter, the conductivity of which would be prevented. The point of interruption must, therefore, be in the gray matter between the terminal

axones of the ganglion cells of the cortex and their union with the ganglion cells. Loewenthal found, on continued impression that no impulse reached the anterior horn, either peripherally or centrally through white substance after two minutes' compression. But the vitality of the non-functionating parts could be restored after anemia of fifteen minutes, which appeared to be the index of their vulnerability. The knee-jerks showed an initial increase which was slowly lost in the same manner as the cortical impulse traveling to the arm and legs. The anemic cord though continued more irritable than normally. The practical demonstration of Hughlings-Jackson's spinal fits is extremely interesting. Slow giratory, writhing movements were present, as a sort of a convulsion long after cortical conductivity to these muscles was abolished by the compression. They were often brought about by peripheral stimulations of stroking the parts in the anemia paralysis. The movements were, for the most part, tonic in character. Probably any motor ganglion may serve under fixed conditions as "cramp centers." But as such groups or centers gradually take higher functions, similar in character to that of the cerebral cortex, they seem to approach nearer to the clonic (cortical) character of true epileptic convulsions. Thus the contention of Jackson in regard to the different level fits receives additional substantiation from this excellent study of Loewenthal's.

4. *Trunk Fits.*—These are some further observations on a case published in *Brain*, Spring, 1902, in which a certain type of status was present. The convulsions began in the muscles of the trunk, i. e., in the muscles of the two sides of the body, the normal action of which may be and often is bilateral and began in those muscles of the two sides simultaneously. In the first period of the status the convulsion did not affect the extremities in the slightest degree. In the second period the limbs were affected but after the trunk. There was a march of the spasm down the arms. The fits were thought to be lowest level fits (ponto-bulbar) and not those of ordinary cortical epilepsy.

5. *Hereditary Aphasia.*—The authors describe a disease that has been observed in eight members of a family; three cases occurring in one generation and five in the generation following. The morbid lesions suggest though far from conclusively the syphilitic origin of the disease. The main features have been; incontinence of urine, attacks of temporary aphasia with loss of power on the right side of the body, gradually increasing opacities in the vitreous humor of the eyes, loss or diminution of the senses by which pain and temperature are appreciated, muscular weakness, epileptiform convulsions, and sudden death preceded by complete unconsciousness. The histories are too detailed for abstracting. The authors have certainly seen a unique type of disease.

6. *External Spinal Pachymeningitis.*—Mills and Spiller report a rare case of this affection in which the entire ventral surface of the dura from the foramen magnum to the caudal end of the dural sheath, was adherent to the bodies of the vertebræ by fibrous proliferations, and the rest of the spinal dura was normal.

7. *Central Cell Changes in Chronic Alcoholism.*—Cole contributes a careful paper to this subject derived from the pathological study of three cases of fatal alcoholic neuritis. His observations suggest: (1) That the peripheral and central lesions express a nervous degeneration of toxic origin, in the production of which no essential part is played by changes in interstitial tissues supporting the nervous structures, or by changes in the blood vessels concerned with their nutrition; (2) The change in the nerve cells are not the mere result of antecedent damage of nerve-fibers, but the changes in the fibers and cells together express a highly selective affection of whole neurones; (3) The peripheral neuritis is simply a local expression of this affection, and is not of purely local and peripheral causation; (4) The lesion of peripheral neurones is only one of many manifes-

tations of the disease, and is accompanied by lesions identical in nature, affecting many groups of neurones situated entirely within the central nervous system; (5) The central changes are not attributable to the peripheral neuritis, and though in some cases the peripheral neurones are mainly affected, in others the morbid process chiefly implicates central neurones; but these two groups of cases do not appear to be sharply divided.

8. *Brain Defects.*—A short note on a defective development of dura mater, old meningitis and internal hydrocephalus occurring in a male idiot of 40 years.

9. *Pain.*—The author defines pain as a disagreeable sensation due to the stimulation of some portion of the cerebrospinal nervous system, and referred to the peripheral distribution in the external body wall of cerebrospinal sensory nerves. Pain in the digestive tract is felt, he believes, across the middle line of the abdomen and in regularly descending areas according as the region affected passes from the stomach to the great intestine. The pain of gastric ulcer is in the epigastrium, the higher the nearer the ulcer to the cardiac end of the stomach. Hyperesthesia and visceral disease he shows to be related, and he further speaks of a viscero-muscle relation whereby visceral disease causes painful muscular sensations, hardness and tenderness of the abdominal muscles in peritonitis being an example. The sensibility of the viscera is an interesting subhead discussed by the author. A thorough reading by the author of Head's researches would have clarified many of his conjectures and hypotheses. JELLIFFE.

### MISCELLANY.

THE PATHOLOGICAL ANATOMY OF CHOREA MINOR. M. Reicharet (Deutsches Archiv für klinische Medicin, LXXI, Nos. 5 and 6, p. 504, 1902).

Two cases of chorea minor are described, which after a brief period resulted fatally, and which upon examination showed anatomical changes in the central nervous system, consisting of inflammation, hemorrhages and degeneration of nerve fibers. The inflammation was characterized by perivenous, diffuse, small cell infiltration of varying degrees; but only in one area was this change visible microscopically. While in several places inflammatory and hemorrhagic changes were found, it is emphasized that the ganglion cells were but slightly affected, if at all. The many hemorrhages, some of which were macroscopical in size, were irregularly distributed, although present throughout the brain substance. These hemorrhagic areas were especially confined to those portions of the brain where the inflammatory changes were most marked, from which the author presumes that the hemorrhages were to be attributed to inflammatory changes in the blood vessels. The location of the fatty degeneration of nerve fibers showed that this change occurred independent of the inflammation, being absent at the central convolutions, as well as in the greater portion of the internal capsule, being present however in a marked degree in a portion of the optic thalamus, in the posterior portion of the internal capsule, in the transverse fibers of the knee of the internal capsule, as well as in the lateral portion of the pulvinar. In the spinal cord, the principal seats of degeneration were the root fibers, fibers in the anterior and lateral cornua, and here and there a fiber in the posterior columns. The anterior and lateral columns remained exempt from degenerative processes. The bacteriological examination of the brain was entirely negative in one case, and at the least doubtful in the other. Worthy of note, however, was the existence of endocarditis and pericarditis, as well as the presence of staphylococcus aureus in the blood within the heart, in one case, and the presence of streptococci in the deposits on the cardiac valves and the heart muscle, in the other case. The author seems inclined to classify these infectious cases of chorea minor with those cases of encephalitis occasionally occurring in the course of other infectious diseases. He attributes the described changes in the brain as a consequence of the influence of toxins. The diffuse dissemination of the anatomical changes in both cases does not permit of a conclusion regarding the origin of the choreiform movements. The cortex, as well as that of the central convolutions, was certainly the seat of only slight changes. The question then arises whether the motor disturbances are to be attributed to the degeneration of nerve fibers which was found to some extent in the basal ganglia and their immediate vicinity. But the author calls attention to the fact, that in from seventy to eighty per cent of cases of post hemiplegic chorea it was in the posterior portion of the internal capsule, in the posterior region of the optic thalamus, or the adjoining portion of the lenticular nucleus, that the lesion existed. DINKELSPIEL (Philadelphia.).

CLINICAL STUDIES AND DEDUCTIONS CONCERNING FAMILY MYOCLONIA AND ALLIED DISEASES. Herman Lundborg.

The author whose name is closely associated with our present knowledge of family myoclonus presents us with a second and more exhaustive monograph of the above title. The author's preface states his study of the conditions dates from 1897. A short historical sketch and a brief résumé of cases by Friedreich, Homen, Unverricht, Sepilli and Bresler are given with the conclusions and the nosological relationship of family myoclonies with the other motor neuroses by different authors. Lundborg gives observations and investigations upon some seventeen cases of family myoclonies from his own clinics, and considers at some length the possible relationship between myoclonia, paralysis agitans, myxedema, Basedow's disease and dementia præcox. He holds that they may develop upon a common defect of the thyroid either structurally or functionally. The monograph is illustrated by photographs and accompanied by genealogic and metabolic tables in original case studies. The author concludes that family myoclonia is a distinct class of the myoclonies, autotoxic in nature, the pathology of which concerns the spinal cord. L. PIERCE CLARK.

UEBER NEUERE KLINISCHE GESICHTSPUNKTE IN DER LEHRE VON DER ARTERIOSCLEROSE. Grassmann (Münchener med. Wochenschr., 1902, No. 109).

The pathological anatomy of arteriosclerosis consists essentially in a diffuse thickening of the intima with a loss of the elastic structure of the media. Hypertrophy of the left ventricle takes place after somewhat advanced degeneration of the visceral arteries or thoracic aorta. The process however, may be localized in the smallest vessels. Persistent arterial tension and deficient nutrition to organs affected characteristic. Arterial strain may be very early recognized and is highly diagnostic as a premonitory symptom.

Syphilis, acute infections, alcohol, toxic substances, such as tobacco in excess, etc., are of etiological importance. Palpation of vessels, at times, of much service in examination. Persistent frequency of pulse on lying down, after erect position, quite significant if accompanied with accentuated aortic second sound. Arteriosclerosis of coronary arteries leads to angina pectoris. When the vessels of lower extremities affected, frequently have intermittent claudication. Small doses of iodide of sodium recommended, with milk diet. Baths of aqua carbonica stated, oftentimes, to have remarkable efficacy.

J. E. CLARK (New York).

ON THE PERMANENT CARE OF THE FEEBLE-MINDED. E. F. Pinsent (The Lancet, Feb. 21. 1903).

In dealing with various plans for the care of feeble-minded persons who naturally fall within the numerous well-defined classes of this condition, the author mentions the following as the last kind of work recently undertaken by the Birmingham After-care Committee. It had been known for some time that there were a large number of idiots, imbeciles, epileptics and feeble-minded persons who had not passed through the regular classes of the institutions, and were not on the ordinary after-care lists of the Committee. With the help of the School Board officials and others, a list of these cases was collected. After a sufficient number had been found, it seemed advisable to appoint a committee, consisting almost entirely of medical men, who undertook to examine and report on these cases. In this way the Committee hopes to inform the City Council of Birmingham the precise extent to which the evil exists. At any rate, it is intended to classify these cases under various heads, which should of themselves make it possible to do something for these dangerous and unfortunate members of society. The classes under which it is proposed to bring these cases are the following, consisting chiefly of two main divisions, namely, patients of the school age and those over the school age. Class "A," those of the school age, i. e., those under sixteen years of age, are divided into (1) Those suitable for special classes for the feeble-minded. These, of course, have already been dealt with by the School Board appropriately, although the medical committee is finding some few cases which hitherto had escaped notice. (2) Cases which for various reasons could be better dealt with in boarding schools for the feeble-minded. Such cases have already been recognized, by the London School Board, for example, and classified under the following heads: (a) Mentally defective children who, but for their defect would be committed to industrial or truant schools. (b) Mentally defective children living in very bad homes. (c) Mentally defective children whose regular attendance it is not possible to secure at any school. (d) Mentally defective children so far from any day school that it would be impossible for them to attend. (e) Defective children known as "morally defective." (3) Epileptics. (4) Epileptics who are also feeble-minded. (5) Feeble-minded children who are also crippled, blind, or deaf mutes. (6) Imbeciles and idiots. The class which includes those over the school age embraces the following subdivisions: (1) Mentally deficient, but capable of industrial work under supervision, i. e., cases suitable for a permanent industrial colony. (2) Mentally deficient, incapable of work, i. e., suitable for a permanent colony. (3) Epileptics who are capable of work in a colony. (4) Feeble-minded epileptics. (5) Feeble-minded persons who are also crippled, blind, or deaf mutes, and (6) imbeciles and idiots. JELLIFFE.

THREE CASES OF INVOLUNTARY MOVEMENTS IN LOCOMOTOR ATAXIA. J. V. W. Rhein, M.D. (Journal Am. Med. Assoc., Dec. 27, 1902).

The involuntary movements of locomotor ataxia may be classified as follows: (1) Associated movements common to many forms of nervous disease; (2) sudden twitchings of the trunk and extremities, occurring with or without pain and more frequently at night; (3) twitchings in isolated muscles or parts of muscles; (4) fibrillary twitchings; (5) rhythmical tremor resembling paralysis agitans; (6) passive movements; (7) involuntary movements commonly described as athetoid movements. To this classification the author adds the rare form of choreiform movements. He describes a case with the classical symptoms of tabes, when a year after the shooting pains began, choreiform movements in the arms, legs and body developed. There was atrophy affecting the anterior and posterior calf muscles and anterior thigh muscles, also in the arms to some extent. There was, in addition to these movements which resembled Sydenham's chorea, a slow involuntary clonic spasm of the fingers of both hands when the eyes were closed. In a second case of tabes, with a spontaneous fracture, and Charcot joint, there was great wasting of the thighs and legs, especially the anterior tibial group. In the left leg a clonic contraction of the toes was observed, which was almost constant. Sometimes the movements were slow, sometimes rapid, but always rhythmical. A third case of tabes with far advanced symptoms showed a curious tremor finely rhythmical in character, resembling the tremor of Parkinson's disease. The pill-rolling posture of the fingers was typically illustrated. There was no rigidity or other signs of paralysis agitans. Discussion of these cases suggested the cerebral origin of the tremor or a combined systemic disease of the spinal cord, not truly tabetic or even disseminated sclerosis. W. B. NOYES.

AN INTERESTING CASE OF PARAPLEGIA WITH RECKLINGHAUSEN'S DISEASE. Maryland Medical Journal, February, 1903.

Patient, female, fifty-one years. No similar condition in family. Always noted certain tumors on her body. Pigment spots also have been observed on skin. Present illness five years' duration. Commenced with stinging pain in ankle, recurring, and gradually extending up leg, involving lower back, then down into other leg. Two years ago similar pains in arms. Commenced to drag one foot. Confined to bed past year, unable to move herself. For some weeks, marked weakness of arms. Jerking of the muscles constant from the first. At times singing in the ears. Dysphasia for past six months, voice now thick and nasal. Catheterization necessary daily. At first no bladder symptoms. Examination reveals great number of tumors, small, from papule to size of cherry, covering body and extremities. Many pigmented areas; also bluish spots. Associated with growths are certain subjective sensory symptoms. The cranial nerves negative; tongue, however, exhibits fibrillary tremors. A condition of muscular atrophy seen in upper extremities. Paralysis of legs, up to and including hip. Patient can move toes of right foot and abduct great toe of left foot. Ankles flaccid. Great atrophy of legs. Deep reflexes of arms retained, those of legs lost. Electrical reactions good except in hand. In the legs only slight response in muscles which are still active. Sensation unimpaired except over tumors. The lesion in this case apparently is extending upward. There is a failure in mental strength. J. E. CLARK (New York).

CONTRIBUTIONS TO THE TUBERCULOUS DISEASES OF THE PONS. Schoeler (Klinische Monatsblätter für Augenheilkunde, XL, No. 2, p. 313, 1902).

The author quotes the histories of three patients, two being adults and one a child, in whom, upon post-mortem examination, tubercles were found on the pons, about the size of pigeons' eggs. In all three cases marked conjugate deviation of the eyes and optic neuritis were found. In one of the patients, who suffered from violent headache, a lumbar puncture was performed; he continued to get worse and died a few days later. According to the author a lumbar puncture should not be performed, if it is assumed that the tumor is deep seated, so as not to cause an increased pressure upon the brain below. After considering the literature on the subject of diseases of the pons, the author states that the characteristic sign of such disease is conjugate deviation of the eyes. Rarely there is limitation of the upward and downward ocular movements. The second characteristic sign is the "Gubler-Millard hemiplegia," the alternating paralysis of the facial nerve and the paralysis of muscles of the extremities or trunk on the opposite side. Ptosis has frequently been found, as well as paralysis of the auditory nerve. DINKELSPIEL (Philadelphia).

THE SURGICAL IMPORTANCE OF INFLUENZA. I. Perez (Deut. Zeitsch. f. Chir., November, 1902).

In a very long article of this title the author draws his conclusions from a review of no less than 520 papers, as well as from his own voluminous experiments on the subject. The more important are as follows: Influenza is a remarkable protean disorder which possesses the highest toxic character, and whose specific cause is the bacillus of Pfeiffer. This bacillus lodges, and causes the characteristic pathologic changes so well known, but often so variable, in almost all the organs, but more particularly in those which, because of the nature of the protoplasm, are most liable to inflammatory processes. The influenza toxins take hold particularly on the central and peripheral nervous system, where they cause either a simple catarrhal process or even purulent, never, however, producing a fibrinous change. The polymorphism of this sickness depends on the location of the infection as well as upon the extent of involvement. Pfeiffer's bacillus enters through the usual paths, viz.: the mucous membrane, the skin, the vascular system, and so on. The course of the sickness depends on the strength of the organism more than upon the path of entry. If the bacilli are introduced in small numbers, they are apt to remain locally. If, however, in great quantities, or if the organism is extremely prostrated at the time of infection, then certain more or less pronounced changes take place, which, in the early stages, present the appearance of a profound toxemia, as a result of which the individual may perish in a short time—from one to three days. On the other hand, however, in the case of a localized inflammation which sometimes appears with great suddenness in several organs at once, there may be present toxic evidences of a subacute nature. In these cases the patient lasts a longer time, from twenty to sixty days, dying finally from marasmus. The influenza bacillus remains for only a short time in the blood stream, being swept by this current into the various organs. It is only on rare occasions that its presence in the blood can be demonstrated. The skin is the most marked region where one expects to find the peculiar changes characteristic of the infection of Pfeiffer's bacillus. They are very varied. In the derma abscess formation is common. In the mouth, the pharynx, the larynx, the trachea and bronchial system the bacillus usually produces a catarrhal inflammation. In the nasal mucous membrane this may be either simple or purulent, and may even progress so far as to form abscesses and hemorrhages. It is in the middle ear, however, that the influenza germ finds its real home. Here it develops and frequently produces empyema of the antrum with the direful sequelæ of meningeal involvement. In the osseous system the bacillus may produce a hypotrophic periostitis as well as suppurative ostitis. In the joints infection of this type usually produces simple serous inflammation, but elsewhere it may become purulent. Even the muscles are not exempt from its inroads, and a suppurative myositis is often met with in the course of influenza infections. In the peripheral nervous system the infection produces degenerative inflammatory changes which may give rise to single or multiple neuralgias or pareses. In the alimentary canal one may frequently find gastro-enteritis, which occasionally proceeds to suppuration, not infrequently producing periappendicular abscesses which too often go on to death from suppurative peritonitis. In the liver a periphepatitis and hyperemia, as well as interstitial hepatitis with necrosis of the liver cells, and possible abscess formation, are to be looked for. In the female generative organs the bacillus of Pfeiffer gives a hemorrhagic endometritis, a salpingitis, a catarrhal vaginitis which may sometimes be purulent. In the ovary alone it appears never to produce any change. In the early months of pregnancy, however, it is prone to produce abortion. An excellent example of symbiosis exists between the staphylococci and Pfeiffer's bacilli, in which the human body is the loser. Any region which has been previously invaded by the first-named germ seems peculiarly suited to harbor the second, and this would appear to be one of the most frequent aids for the entry of the influenza germ. In establishing a diagnosis of influenza infection, the microscopic investigation of the discharge is of the highest worth, and it is characteristic that the bacillus is found almost always within the cells. The serum taken from animals infected with influenza produces a moderate agglutination reaction, but this is not sufficiently constant to be of marked diagnostic value. One important differential diagnosis to be made is whether a given case is pure influenza infection or contaminated with the pneumococcus, this combination being frequent and virulent. There is no such thing as passive immunity for influenza, only a moderate and rapidly vanishing active immunity seeming to be produced. It will be seen that many of these conditions, which should now be recognized as due to Pfeiffer's bacillus, are too often classified under the old and unscientific name "rheumatism." JELLIFFE.

SURGICAL TREATMENT OF HEMORRHAGIC PACHYMENINGITIS. John C. Munro. (The Chicago Medical Recorder, December, 1902).

The conclusions of the author, in a very interesting and instructive paper presented at the October meeting of the Chicago Medical Society, are (1) Hemorrhagic pachymeningitis is found in the insane and in infants, but for the most part in men not insane after middle life; (2) Alcohol, syphilis, acute and wasting diseases, and trauma apparently bear some causative relation; (3) The symptoms are those of diffuse subdural hemorrhage, coming on slowly, producing mental irritation, spasm and rigidity of the extremities, convulsions, and later, paralysis, the sequence being more or less irregular; (4.) The cranial nerves are not liable to be affected; (5.) The treatment is surgical, and should be instituted as early in the disease as possible. (6.) Without operative relief in cases with pressure symptoms, the prognosis is practically hopeless. Early operation is not serious and gives the best chance for recovery.

J. E. CLARK (New York).

SUBDURAL INTERPOSITION OF RUBBER TISSUE WITHOUT REMOVAL OF THE GASSERIAN GANGLION IN OPERATIONS FOR TIC-DOULOUREUX. R. Abbe (Ann. of Surg., Jan., 1903).

The hemorrhage and the dangers from hemorrhage and shock led the author to reason that mere division of the affected trunk of the nerve within the skull, coupled with the interposition of a bit of rubber tissue between the divided ends would not only obviate the major operation of removing the ganglia, but also effectually prevent the reunion of the cut nerve. The operation which he advocates in grave cases of this disease is that the surgeon should not temporize by any of the external methods of operating, but at once resort to this, which now seems to him the proved and radical cure in its safest form. The external carotid artery may be ligated with advantage in controlling hemorrhage. A vertical incision over the middle of the zygoma carried through the temporal muscle to the bone divides no important nerve or vessels. The muscle is scraped to either side and held by retractors. A small opening is quickly made by mallet and gouge, and this is rapidly and safely enlarged to an inch and a half diameter. No better exposure can be had by any incision than this simple straight one. The dura is then pressed away from the middle fossa until the nerves are exposed. The much complained hemorrhage from venous sinuses on dissecting up the periosteum can be best controlled, and very quickly, by pressing a strip of rubber tissue upon the place with a firm pad of gauze in strips. The clotting of blood under the rubber tissue takes place very quickly, while if plain gauze is put in contact with the bleeding point, the blood being sucked up into it, prevents clotting. The nerve trunks he grasps in separate artery clamps, divides each close to the foramen of exit, and, either by cutting or by rotation of the forceps, separates them from the Gasserian ganglion. The wound is packed for a few minutes with narrow strips of iodoform gauze until dry. A piece of thin gutta-percha tissue, stiff enough to be easily handled, is sterilized by rubbing with bichloride solution, and kept in salt solution for a few moments before operating. This is cut 1½ inches long and three-fourths of an inch wide. This is laid carefully over both the foramen rotundum and ovale, where the nerves have been separated and pressed carefully into place by iodoform gauze. In a very few moments the gauze may be drawn away, and the Gasserian ganglion allowed to settle down upon the rubber tissue. A small drainage-tube should be placed in the angle of the wound to insure a perfectly dry healing. It certainly is beyond dispute that there is no need for the removal of the first branch of the fifth pair in any case of grave tic douloureux unless the origin is to be found in a tumor of the Gasserian ganglion or behind it. In conclusion, he says that he thinks that he has demonstrated (1) that the operations upon the ganglion have been carried to an unnecessary degree of severity; (2) that resection of one-fourth or one-half inch of the nerves anterior to the ganglion and within the cranium, with the interposition of rubber tissue, can be relied upon for perfect cure, up to six years at least, with probability of permanency as great as by any method; (3) that it is a simple, speedy, and safe method, and thereby fulfils the highest aims of the best surgery. JELLIFFE.

THE CHANGES IN THE SPINAL CORD AND MEDULLA IN PERNICIOUS ANEMIA. Frank Billings (The Chicago Medical Recorder, Jan., 1903).

From careful study of the subject the author concludes (1.) That there is a well established relation of diffuse cord degeneration with pernicious anemia; (2) It seems probable that the hemolysis and the cord changes are due to the same toxin; (3.) While the source of the toxin is unknown, the fact that gastro-intestinal disturbance is so common in the disease would lead one to suppose that it is of intestinal origin; (4) The diffuse degenerations of the spinal cord which occur in conditions without pernicious anemia do not appear to differ essentially from those of pernicious anemia; (5) It is possible that a common blood circulating poison exists which may expend its force upon the blood in one individual, upon the nervous apparatus in another, and coincidently upon the blood and spinal cord in others.

J. E. CLARK (New York).

HEREDITY OF MENTAL DISEASE IN GENERAL. Dr. J. Wiglesworth, Medical Superintendent of Rainhill Asylum (The Hospital, Feb. 14, 1903).

The author deals at some length with important and practical facts collected in a long experience and referring to the question of the hereditary transmission of mental diseases. It used to be taught in the earlier days of biological science that "the respective shares which the sperm-cell (spermatozoon) and the germ-cell (ovum) contributed to the act of fertilization were of a different order, the germ cell furnishing the matter, the sperm-cell supplying the force which animated that matter, and started it upon its career of development." This view can no longer be held in the light of modern research. We now know that the portion of the germ-cell or ovum which takes the main part in the formation of the fetus or offspring is the nucleus. It is to the union of the nucleus of the sperm-cell with that of the germ-cell that the development of the fetus is due, the two nuclei (male and female respectively) contributing eoual shares, in the material sense, to the process. The nuclear material which thus constitutes the vehicle of transmission of herditary qualities to the fetus from both parents, is itself an organized and complex structure, into whose composition there enter probably many millions of molecules of multiform character, and which singly or in groups take part in the development of every portion of the fetus. Prior to fertilization the nucleus of the ovum undergoes a series of changes whereby one-half of the nuclear "chromatic substance" is divided off and extruded. Precisely analogous changes take place with regard to the nucleus of the spermatozoon with the exception that each half of the "chromatic substance" resulting from the division continues as a sperm-cell. Although the precise interpretation of these "reducing divisions" is still obscure, the net result is obviously to diminish by one-half the chromatic substance contained both in the germ-cell and in the sperm-cell. And it seems clear that the chief object of this process is to prevent indefinite increase in the mass of the nuclear substance, and thus to secure uniformity of mass. But this "reducing division" is not merely a process for keeping the mass of the nuclear substance uniform in size. For prior to the operation of "reducing division" the nuclear structures (chromatic rods) undergo a process of splitting or cleavage by which each chromatic rod is split longitudinally into halves. Weismann, whose monumental work on heredity has done much to illuminate the subject, believes that the object of this primary doubling of the nuclear rods is to increase the possible number of combinations of the germ plasma, and therefore the number of varieties which result from the union of the parent cells after "reducing division" has been completed. This is clearly a factor of immense importance in phyletic development. Natural selection acts always and everywhere by seizing upon and fixing favorable varieties, and the greater the number of variations it has to work upon, the greater is the chance of a type being developed adapted to any particular environment. There can be no doubt that only a fraction of the countless variations which are being continually produced become permanently fixed, the rest being silently quenched by the operation of this great natural law. Hence the vast importance of obtaining the greatest possible diversity in the minute structure of the germ plasma, so as to furnish an abundant material for natural selection to work upon. The above observations will show that the problem of heredity is one of great complexity.

The problem of heredity is for the alienist one of particular interest and importance, for it is daily demonstrated that mental (cerebral) peculiarities, no less than bodily ones, descend from parent to child, and that the aberrant mental traits of one generation are the logical sequence of the mental abnormalities of preceding generations. The practical questions which may be addressed to psychological medicine on this point are many. For example, what proportion of insane parents owe their insanity to definite hereditary taint? and in how many, on the other hand, can the insanity be said, in the strictest sense, to have been acquired in the course of the individual life? Is one sex more prone to hereditary insanity than the other? Is one parent more potent than the other in transmitting the disease to the offspring, and are the male and female children affected in different numerical proportions, according as their insanity is derived from the father or the mother? Do the different forms of insanity differ from one another in the degree with which they tend to be inherited? Can the result of disease acquired by a parent be transmitted to the offspring so as to appear in the latter in the form of a tendency to insanity? Is there any diathesis (other than insanity) occurring in one parent which, when associated with the insane diathesis in the other parent might tend to neutralize transmission of the latter diathesis to the offspring, and, conversely, is there any diathesis which tends to reinforce the insane diathesis and to add, therefore, to the dangers? With the object of answering some of these questions an inquiry was made into the family history of a series of cases of insanity observed by Dr. Wiglesworth. These statistical inquiries deal with a series of 3,445 patients admitted to Rainhill Asylum during a period of 12 years, 1,693 of the patients being males, and 1,752 females. These were "patients concerning whose family history any details whatever were obtainable," and in whose direct parentage (fathers, mothers, and grandparents) or collaterals (brothers, sisters, uncles and aunts) insanity, if it was met with, was regarded as a hereditary factor. Patients whose cousins only afforded evidence of insanity were not included in the class of hereditary insanity, as it was thought that there was an equal chance of such taint having been introduced by marriage from an entirely different family, and it could not be assumed, without further proof, that such taint belonged to the family of the patient. "Out of the whole series of 3,445 patients a definite hereditary taint of insanity, epilepsy, or a marked degree of eccentricity or peculiarity, either direct or collateral, was found in 965 cases, a percentage on the whole number of 28.01." There can be no doubt that this figure errs, adds Dr. Wiglesworth, on the side of deficiency, as it is difficult to get trustworthy information as to the family history of our patients, especially when they belong to the pauper class. Farquharson, a recent observer, dealing with a large number of patients of a similar class, obtained a percentage of hereditary taint of 30.7. Statistics drawn from institutions which receive private patients—as distinguished from paupers—usually give appreciably higher results. Thus Grainger Stewart found a percentage of 49.6 among the patients admitted into the Crichton Royal Asylum. Reverting to Dr. Wiglesworth's figures, which give an hereditary taint of insanity in 28.1 per cent, or 965 out of 3,445 patients, it is found that the sexes exhibit a difference. Of these 965 patients, 419 were males and 546 females; and a simple calculation of these numbers in relation to the separate totals of male and female patients investigated in the report shows that the percentage of hereditary taint was 24.74 for male patients, and 31.16 for females. This striking difference has an interest which is enhanced by the fact that it harmonizes with the records of previous investigators, almost all of whom have found a higher percentage of hereditary taint among the female patients than amongst the male. Thus Thurnam gives a percentage of 32.82 for the males and 35.48 for the females; Grainger Stewart gives 48.56 for males and 51.05 for females; while Farquharson gives 27.4 per cent for males and 34.16 per cent for females. The conclusion has hence been drawn that the female sex has a greater liability to suffer from hereditary insanity than the male. It has been suggested that the female sex has to pass during life through physiological crises—puberty, pregnancy and the parturition, lactation, and the menopause—which constitute periods of danger, and which are prone to evoke into activity any latent hereditary taint of insanity, whereas in the male sex such danger is over. when the age of sexual maturity (25 years) is attained. It appears, however, that certain forms of insanity, to be mentioned later, are more often acquired in the lifetime of the individual in males than in females, and that when these are excluded the difference between the sexes as regards the hereditary incidence of insanity it not so marked.

How Not to Be Nervous. Hugh T. Patrick (Journal American Medical Association, February 7, 1903).

For preventing nervousness in the child or removing that already present, nothing is so effective as the toughening of the body and mind. A child with hard muscles, strong lungs and a vigorous digestion, who can bear changes of temperature and endure pain is already a long way from nervousness. More important is the "toughness of the psychic fiber." The child who can support disappointment, who can be "crossed" without a tantrum, and who habitually obeys is building a bulwark against "nerves." Beside premature entrance into society and magnifying the importance of a child in the family, harm is done by allowing the child to experience fear, usually started by suggestions of parents, or nurses. The writer shows that overwork seldom causes nervousness, but the lack of work, misplaced energy, or worry and indecision surely causes it. Overrating one's natural ability and failure to appreciate the limit of intellectual and physical power surely develops nervous conditions sooner or later. W. B. Noyes.

The Diagnostic Symptoms of Tumors of the Brain. J. Arthur Booth (Medical Critic, February, 1903).

Of the general symptoms of brain tumor, headache, vomiting, vertigo, mental dulness and optic neuritis, headache is the most frequent and distressing; but unless associated with other general symptoms it is not of much diagnostic importance. Localized pain with tenderness on pressure suggests that the tumor is superficial. Vomiting without obvious cause at irregular intervals is a common and distressing symptom. Vertigo is common, especially in lesions of the cerebellum or base of the brain. Double optic neuritis is the most important symptom, without which a positive diagnosis is difficult. The group of localizing symptoms is important, especially motor symptoms. Of cases reported by the writer, the first at operation showed a large superficial sarcomatous mass at the upper part of the left anterior central convolution. This had caused double optic neuritis, epileptic attacks, the spasm at first limited to the big toe of the right foot, but later involving the arm and leg of the same side; no loss of consciousness; blurring of vision and later blindness of the left eye. Headache and vomiting were late symptoms.

In the second case an operation yielded a sarcoma in the facial center of the left motor zone. The symptoms had been pain in the head, and recurring spasmodic twitchings of the right cheek and neck, not involving the arm. Speech was wholly abolished at such times. Later the right hand became somewhat affected. Paralysis of the lower facial muscles of the right side was developed; double optic neuritis; increased knee jerk on the right side; pain and tenderness over the motor zone.

In the third case, a mass about the size of a hen's egg was removed from the left frontal lobe. This had caused attacks of general convulsions and unconsciousness; progressive failure of vision, severe headache, chiefly in the temples; poor memory and other mental symptoms; no special paralytic symptoms; double optic neuritis; deficient sense of smell on the left side, a prominent and tender swelling just back of the external angular process of the frontal bone.

The fourth case at autopsy revealed a large round cell sarcoma, completely filling the right ventricle and extending over into the left. The corpus callosum and fornix anteriorly were invaded. The symptoms had been a persisting frontal headache, morning vomiting, failure of vision and double optic neuritis; equilibrium poor, with a tendency to fall to the left side. Visual illusions due to a varying hemianopia were noted. Later, fainting or epileptic attacks, loss of sense of smell, paralysis of the left external rectus, diminished hearing on the left side; left hemiplegia and blindness. Mental stupidity, coma and death sixteen months from the onset.

In the fifth case, at autopsy a large nodular growth three inches long was found extending into the right lobe of the cerebellum. This extended also into the fourth ventricle, pushing the medulla to the left and pushing on the right crus of the cerebellum. The symptoms had been progressive malaise, headache, recurring vomiting, mental dulness, irritability, stiffness of the neck. Slight paresis of the right external rectus, no reaction of the pupils to light, marked sensitiveness to touch all over the head. Later optic neuritis and well-marked convulsions; dimness of vision. Five months later complete blindness, semi-coma, paralysis of right side of face, partial paraplegia, bulbar symptoms. W. B. NOYES.

EPILEPSY. Its Psychopathology and Medico-Legal Relations. H. A. Tomlinson (Journal American Medical Association, Jan. 17, 1903).

The more rapid the mental reduction after the establishment of the convulsive habit the greater the psychic perversion and the more frequent those manifestations of mental aberration which belong to the epileptic, the post-convulsive furor and automatism. The study of these maniacal outbursts indicates that they are the response to the impelling suggestions of unreasoning fear, following vivid hallucinations, usually visual but sometimes auditory. Or, in cases where there is much mental reduction, it is the expression of brutal impulse to wanton cruelty in response to the gross irritation of the convulsive seizure. Sometimes these outbreaks, especially in the procursive form, are mere "running amuck" without regard to anything which comes in the way and with no purpose. Epileptic automatism implies a less degree of mental reduction and the persistence of considerable intellectual capacity. The persistence of automatism after the convulsion constitutes what is known as double consciousness. At such times a return to original or concealed traits of character or family traits will occur. The term psychic epilepsy has no real significance because it implies a condition which, in the nature of things, could not exist; that is, that there is a mental instead of a motor convulsion. It is almost certain that this condition is a post-epileptic phenomenon, where the initial attack is petit mal, which is observed by the mental aberration which follows and is manifested by the automatism or maniacal outbreak. Therefore it is the sequence to the explosion and not its equivalent. In determining how far the epileptic may be responsible for his acts we have to consider not his epilepsy but the degree of primary defect in his mental make-up as manifested by his cerebral potentiality and intellectual capacity. An epileptic child is more or less "spoiled" and poorly controlled. When out in the community, not getting the same treatment, morbid self-consciousness, resentment even to paranoia may develop; or outbreaks of excitement, mental depression or delusions. W. B. NOYES.

THE MINUTE ANATOMY OF ERYTHROMELALGIA. H. Batty Shaw (British Medical Journal, March 21, 1903.)

Erythromelalgia is the name given to a condition of the extremities in which redness and pain are associated. Dependence produces considerable increase of the dusky red or violaceous tint of the extremity affected. The pain is increased by warm rather than cold weather; there is no loss of sensation, but there may be increased sensitiveness. The affection is asymmetrical; gangrene does not occur; muscular wasting is found, but is caused by disuse and is less severe than in neuritis. A reaction of degeneration in the nerves of the affected parts has not been found. The deep reflexes are not reduced. In examination of amputated fingers in three cases of erythromelalgia the following results were obtained: The digital arteries showed very slight thickening; those of the thumb showed obvious thickening in the internal coat. There was no definite sign of degeneration of any of the nerves, nor was there an apparent deficiency of nerve fibers or any increase of fibrous tissue in the larger nerve trunks or in the digital nerves. No syphilitic history could be obtained or signs of central nervous disease. Of nine other cases reported a reduction of nerve fibers in a portion of nerve excised was found with an increase of fibrous tissue. W. B. NOYES.

# Book Reviews.

Les Obsessions et la Psychasthenie. Fragments des Leçons Cliniques du Mardi sur les Etats Neurasthéniques, les Aboulies, les Sentiments d'Incomplitude, les Agitations et les Angoisses Diffuses, les Algies, les Phobies, les Delires du Contact, les Tics, les Manies Mentales, les Folies du Doute, les Idées Obsédantes, les Impulsions, leur Pathogenie et Leur Traitement. Par le Dr. F. Raymond, et le Dr. Pierre Janet, Paris, Felix Alcan, 1903.

This is the fourth series of observations from the laboratory of psychology in the nervous clinic at the Salpêtrière. Professor Janet, the distinguished Professor Ribot's favorite pupil in psychology and himself one of the best known of living psychologists, has added a distinctly new element to Professor Raymond's well known work in the nervous department of the old Salpêtrière hospital. The present volume is indeed an informing one. When an American introduced the word neurasthenia, only about a quarter of a century ago, neither he nor any one else had any idea of the number of symptom-complexes of various kinds that would be described, the basis of which would prove to be a neurasthenic condition. Not all of these often very queer conditions are mentioned in this volume. Yet its five hundred odd pages contain descriptions of the most diverse mental states associated with neuroses differing not only in kind but in degree, through a whole scale of variations.

It used to be said that hysteria was a disease that could simulate almost any other nervous disease. Now we know that under the term major neurosis and therefore in a way justifiably to be called hysterical there are a variety of symptoms almost without end. The affections partake of a combination of mental and physical symptoms that are almost inexhaustible in their individualizing details. Pains and fears and anguishes and phobias with manias for touching and for fearing contact, for avoiding and for repeating, with manias of doubting and of compulsion, are all almost inextricably associated together without any definite modality in a way that shows the inexhaustible quality of nervously disturbed nature.

After reading Professor Raymond and Janet's book it is easy to understand how the quack and the charlatan and the Christian Scientist find material to work on. There are endless nervous symptoms for which the most effective treatment is suggestion in some form with improvement of the general nutrition. If for no other reason but the recognition of this fact their book deserves to be widely read by the general practitioner as well as the nervous specialist.

J. J. Walsh (New York).

# News and Notes.

**Dr. Merzbacher** has been appointed as Assistant Physician to the Psychiatric Clinic in Freiburg, Baden.

**Professor von Strumpell** has been invited to occupy the position as Director of the Medical Clinic at Breslau.

**Dr. L. Bruns** of Hanover has been made Professor.

**At the last annual** meeting of the German Alienists held at Munich, a commission was appointed to collect statistics bearing on all criminal acts perpetrated by the insane. Cases of suicide, homicide, litigation, sequestration of the insane, insane soldiers and their relations to superior officers, political cranks, etc.

**The Ninth International** anti-alcoholic congress was held at Bremen April 14, 19 last.

**Prizes Taken by Neurologists and Alienists**: Prizes of the Academy of Medicine of Paris for 1902 were awarded as follows: Baillarger Prize to M. Pactel and H. Colin, on the Insane in Prisons and the Insane before the Courts; Chas. Boullard Prize to M. P. Calalian on Hypochondria in Insanity and the Neuroses; T. Herpin Prize to M. R. Cestan on Little's Syndrome; Laborie Prize to M. Jaboulay on Surgery of the Nervous Centers.

**Academy of Science** of Paris Prizes were awarded to M. Dejerine for his Diseases of the Nervous System; Mlle. Pompilian and M. G. Hauser, for their Study of Syringomyelia and to M. P. Bonnier for a Study of Orientation and the Sense of Altitude.

**Jacopo Finzi** of Florence, a rising young alienist, died at the age of twenty-nine of typhoid fever. A notice of his achievements is given in the last number, No 1, of the Giornale di Psichiatria clinica.

**American Neurological Association.**—The program of this Society, holding its next annual meeting at Washington, May 12, 13, 14, is as follows: Tuesday, May 12, presidential address, Dr. J. W. Putnam, of Buffalo. Other papers are: The Sign of the Orbicularis in Peripheral Facial Paralysis, by Dr. George W. Jacoby, of New York; Contribution to the Study of the Achilles Reflex and the Front Tap, by Dr. George L. Walton and Dr. W. E. Paul of Boston; The Reflexes in Long-distance Runners, by Dr. Philip C. Knapp and Dr. J. J. Thomas, of Boston; Report of a Case of Aneurism of the Descending Thoracic Aorta Mistaken for Pott's Disease, with Autopsy, by Dr. Charles W. Burr, of Philadelphia; Polioencephalomyelitis and Allied Conditions, by Dr. Edward W. Taylor, of Boston; A Case of Acute Degeneration of the Spinal Cord with an Ascending Type of Paralysis, by Dr. D. J. McCarthy and Dr. Wm. E. Hughes, of Philadelphia; Two Cases of Multiple Tumors of the Brain and Spinal Cord, and One Case of Primary Sarcoma of the Spinal Cord, by Dr. William G. Spiller and Dr. William F. Hendrickson, of Philadelphia; Three Cases of Spinal Cord Tumor Treated by Operation, by Dr. J. J. Putnam and Dr. J. W. Elliot, of Boston ;Two Cases of Brain Tumor, with Operations. Recovery and Ultimate Death, by Dr. C. Eugene Riggs, of St.

Paul; A Cerebellar Tumor; Operation; Recovery, by Dr. Frank R. Fry, of St. Louis; Central Neurofibromatosis: A Contribution to the Study of Tumors of the Medullo-Ponto-Cerebellar Angle, by Dr. Joseph Fraenkel and Dr. James R. Hunt, of New York; A Case of Progressively Developing Hemiplegia, Later Becoming Triplegia, Resulting from Degeneration of the Pyramidal Tracts, by Dr. Charles K. Mills and Dr. William G. Spiller, of Philadelphia; Chronic Progressive Hemiplegia, by Dr. Hugh T. Patrick, of Chicago; A Case of Progressively Developing Left Hemiplegia, Followed by Progressively Developing Right Hemiplegia, with Exhibition of the Brain, by Dr. Graeme M. Hammond, of New York; Flaccid Paralysis Following Cerebrospinal Meningitis, by Dr. William N. Bullard, of Boston; Cases of Alexia, by Dr. Philip Zenner, of Cincinnati; Two Cases of Hysteria Presenting Marked Symptoms of Insanity, by Dr. Theodore Diller, of Pittsburg; The Principles Underlying Practice in Mental Therapeutics and Legal Regulations of Its Practice, by Dr. Richard Dewey, of Wauwatosa; The Neurasthenic Neuralgias, by Dr. Frank K. Hallock, of Cromwell; The Intracranial Complications of Middle-ear Suppuration, by Dr. Wm. M. Leszynsky, of New York; Modern Theories of the Emotions and Their Bearing on Mental Derangements, by Dr. Henry Upson, of Cleveland; Paralysis Agitans Complicated with Delusional Mania, by Dr. John Punton, of Kansas City; Atypical (One-sided) General Paralysis, by Dr. Adolf Meyer, of New York; Report of Two Cases of General Paralysis with Focal Symptoms; Autopsy in Both Showing Hemiatrophy of the Brain, by Dr. A. Hoch, of Waverley; The Facial Reflexes, by Dr. Charles L. Dana, of New York; The Importance of the Plasma Cell in the Cortical Vessels in Dementia Paralytica, by Dr. Stewart Paton, of Baltimore; A Case of Cerebral Tumor in which Muscular Atrophy and Astereognosis were Prominent Symptoms, by Dr. Morton Prince, of Boston; Bony Sensibility by Dr. Philip C. Knapp of Boston; Intense Double Optic Neuritis Without Assignable Cause, by Dr. Howell T. Pershing, of Denver; Remarks on Astereognosis, by Dr. William N. Bullard, of Boston; The Importance of the Nerve Centers in Pulmonary Consumption, by Dr. Thomas J. Mays, of Philadelphia; The Limits of Non-physical Therapy, by Dr. Smith Baker, of Utica; Demonstration of Certain Points Regarding the Development of the Neurofibrils in the Nerve Cell, by Dr. Stewart Paton, of Baltimore; Cases of Poisoning with Morphine and Atropine, by Dr. Philip Zenner, of Cincinnati; Two Cases of Insular Sclerosis, with Demonstration of Specimens, by Dr. J. J. Putnam and Dr. E. W. Taylor, of Boston; A Case of Lower Arm Type of Paralysis Dating from Birth, by Dr. John J. Thomas, of Boston; The Cerebral Origin of the Knee-jerk, by Dr. J. W. Courtney, of Boston; On a Cervical Type of Spinal Syphilis Closely Resembling Syringomyelia, by Dr. B. Sachs, of New York; Paralysis Agitans in Early Life, Complicated by Symptoms Suggestive of Multiple Sclerosis, by Dr. B. Sachs, of New York; The Work of a Large Dispensary Clinic, by Dr. Smith Ely Jelliffe and Dr. L. Pierce Clark, of New York; A Case of Primary Sclerosis of the Posterior Columns, Followed by Disseminated Softening of the Other White Columns, by Dr. Joseph Collins, of New York; Disseminated Myelitis, by Dr. Joseph Collins of New York.

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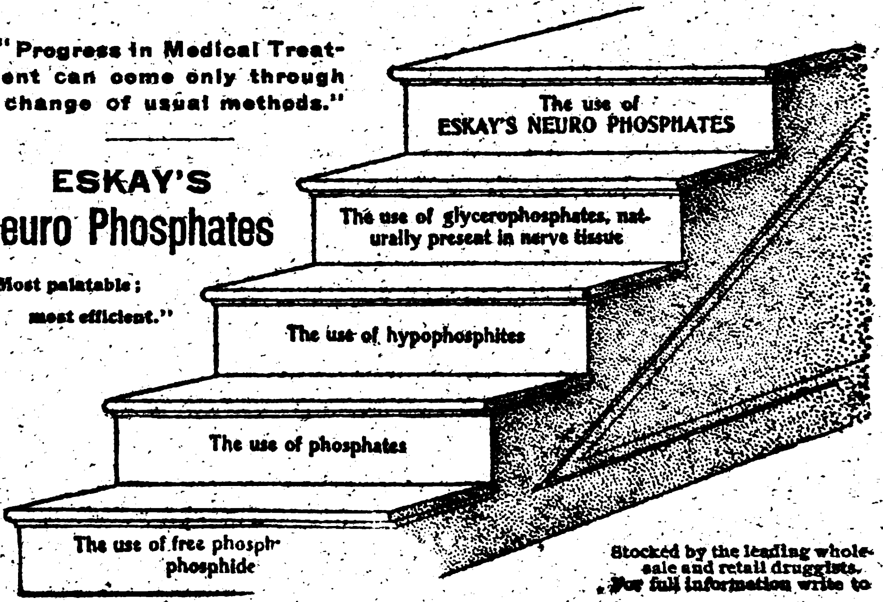
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Vol. 30 June, 1903 No. 6

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The Official Organ of

The American Neurological Association
The New York Neurological Society
The Philadelphia Neurological Society and
The Chicago Neurological Society

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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

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Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

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ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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# THE Journal OF Nervous and Mental Disease

## Original Articles.

### PRESIDENTIAL ADDRESS.[1]

By J. W. Putnam, of Buffalo, N. Y.

Gentlemen—Before proceeding to address you formally, I desire to express my warmest thanks for the very great honor you have conferred upon me in choosing me for your twenty-ninth President. With the honor I accept also the responsibilities, and rely upon your help in making this a successful meeting.

The American Medical Congress was founded because the interdependence of the different branches of medicine was rcognized by the master minds of the profession. The time had come when specialists needed to broaden their interests and enlarge their views of the field of medicine. Specialism is so engrossing that its devotees are in danger of becoming narrow in their interests and their activities.

Of no class of physicians is this more true than of neurologists. The range of diseases we are called upon to treat is so large and so varied that it would seem impossible for us to become narrow. In the past few years we have been especially active in research along many lines. Our text-books are models of exact and definite descriptions of diseases, of careful observations of symptoms, of detailed records of clinical history, and of the minutiæ of pathological findings.

Though our writers are accurate and complete in their observations there is an evident tendency to specialize in our therapeutics. Our prognoses reveal to some extent the natural tendency of diseases. Our view of the future seems to be limited by our own resources.

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[1] Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

It is important that as teachers and authors we bear in mind that the treatment of disease resulting in benefit to the patient is the ultimate goal of our efforts. It is to this end that our hospitals are founded and maintained by the charitable; it is for this purpose that our patients seek us, and for this that students read and study our books.

The chronic cases of paralysis with deformities from various cerebral, spinal and peripheral causes form a large and hitherto hopeless class. For years those afflicted with diplegia, hemiplegia and spastic paraplegia have sought medical aid in vain. In the books on nervous diseases which the physician consulted for guidance he found, until recently, at the best a statement that such a case may be benefited by appropriate mechanical appliances, massage and electricity; he seldom found that definite statement of facts which the orthopedists have accumulated in the last decade.

Tendon transplantation and the principles which govern the operation should find a place in our chapters on treatment. It should be taught to the student from the neurological standpoint as well as from the surgical. The field is a new one. Old methods are giving place to better ones. This is a therapeutic measure which appeals specifically to us, for it is designed to benefit our patients. It is our province to develop this practice; to study, investigate and teach it, to consult with the surgeon on special cases as we do in a case of brain tumor.

We have long ago made common cause with the surgeon in cerebral and spinal operations; let us extend it to the domain of surgery of the neuromuscle machine.

To the surgeon belongs the technic, and this has been ably developed by such operators as Bradford, Gibney, Goldthwaite, Townsend and Whitman of this country; by Robert Jones and Tubby in England; Hoffa, Lange and Vulpius in Germany; Codivilla and Niccoladoni in Italy.

To us belongs the duty of selecting cases for operations and of enlarging its field of usefulness. The after-care of the patient is as important as the surgical. The operation must be followed by an education, both mental and physical, which we must direct. It is only through coöperation that the best results will be obtained.

Hitherto the relation of neurology to obstetrics has not been sufficiently appreciated nor emphasized. The effect of prolonged labor upon the child is being studied, and requires much more careful investigation in order to determine the effect of cranial injuries upon the brain. The obligation rests with us to sound the note of warning, in season and out of season, that epilepsy is in a large number of cases due to cranial birth lesions. The lesson must be so impressed that it will be a rule with accoucheurs to examine the head for fractures or undue depressions after severe labors. When this practice has become a rule, and the cranial injuries are treated early and carefully, we may see as a result a diminution in epilepsy, in idiocy and cerebral birth palsies.

It has not been my purpose to refer to the most excellent work done in our own lines of investigation, so much as to emphasize the fact that specialists in the other societies are developing principles and accomplishing results which are of vital importance to us, and that we are developing theories and obtaining facts of equal value to them, and that by joining our researches and making a united effort in investigating the causes of diseases and new methods of treatment, we may improve somewhat the helpless condition of those for whom the prognoses have hitherto been unfavorable, and in other instances diminish in some measure the deplorable results which are to be found in our homes for the epileptic, the feeble-minded and the incurable paralytics.

# TABES AND MUSCULAR ATROPHY.[1]

BY JOSEPH COLLINS, M. D., NEW YORK.

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE POST-GRADUATE MEDICAL SCHOOL; VISITING PHYSICIAN TO THE CITY HOSPITAL;
CONSULTING NEUROLOGIST TO THE HOSPITAL
FOR RUPTURED AND CRIPPLED.

Some degree of muscular atrophy is not uncommon in tabes, in fact such complication occurs in from 15 to 20 per cent of all the cases. It is responsible for many of the deformities of the feet which occur late in tabes, such as the tabetic clubfoot and sometimes for the total motor incapacity amounting to paralysis. The variety or form of atrophy that I shall consider particularly in this article, however, is not the slighter forms that lead to deformity of the feet and hands. I shall consider rather the muscular atrophy that is occasionally encountered with tabes, which resembles in its distribution and course progressive muscular atrophy of spinal origin. The other forms of progressive muscular atrophy have recently been described by me in an article in the *Medical News* dealing with the Symptomatology of Tabes. The variety of atrophy of which the cases that I publish herewith are examples has nothing peculiarly characteristic of it in its clinical manifestations, so far as I can make out, save that it overshadows the tabes, which is rarely typical, in its clinical delineation. The atrophy may attack any part of the body, and extend so as to involve all of the extremities, as it did in my first case, or it may confine itself entirely to the upper extremities, as it did in my second case, or it may be localized, as in my third case. Occasionally, amyotrophy in tabes, which is probably not unlike in its causation the form that I am describing, is limited to a smaller area than in the third case, for instance, the half-sided atrophy of the tongue that was described by Charcot as a complication of tabes may be of the same origin, and likewise the atrophy of the soft palate that has been mentioned by Marie and Koch.

When the extensive amyotrophies of tabes were first described by Condoleon, Leyden, Charcot and Pierret, it was generally accepted that they were dependent upon alteration of the anterior horn cells, either parenchymatous decay or diminution in number. Some of the cases reported by these authors, as well as those of Chretien and Thomas, prove that tabetic amyotrophy is in some instances dependent upon cytological changes in the anterior horn cells. On the other hand, during the past fifteen years a very considerable number of cases have been reported in which disease of the anterior cornual cells has not been found. The muscular atrophy in these cases is adequately explained by the lesions of the peripheral nerves that accompany them. So today, we must admit that amyotrophy of a very profound type occurs with tabes, and that it is dependent in some instances upon lesions of the anterior horn cells, in others, the more frequent, upon disease of the peripheral nerves. It is true that in some cases of extensive muscular atrophy in tabes we have, as Dejerine has suggested, a coincidence of tabes with chronic poliomyelitis, just as occasionally we have it with other chronic nervous disease, such as paralysis agitans (Placzek), and multiple sclerosis (E. W. Taylor). I see no reason why a patient with tabes should not develop paralysis agitans, or multiple sclerosis, but of course, there is no relationship between them. Slight alteration of the peripheral nerves is not infrequently found when the lesions of tabes are studied with the microscope[2] and a theory of the pathogenesis of tabes has been founded in part upon the existence of these lesions. The changes in the peripheral nerves in tabetic amyotrophy are of a different nature from those found in tabes with slight muscular atrophy, and the lesions of the former cannot be looked upon as an intensification of the process in the latter. As a matter of fact, the slight alterations of the peripheral nerves that have been described in some cases of tabes may occur in almost any slowly progressive central nervous disease of protracted duration. In the severer forms, such as Case I in this series, the lesion is a true parenchymatous inflammation or degeneration. Some writers have endeavored to make the changes in the peripheral nerves secondary to functional affection of the anterior horn cells, for instance, Schaffer[3] maintains that the anterior horns are functionally damaged consecutive to degeneration of the collaterals of the posterior roots. The cellular affection begins with very delicate structural changes which develop slowly. The clinical character of tabetic atrophy is in complete harmony with the cellular changes. The most characteristic feature is its slow progression, the absence of fibrillary twitchings and of reaction of degeneration. The most common location is in the peroneal region. Schaffer records a case in which Argyll-Robertson pupil, loss of the patellar reflexes on the right side, and a tabetic arthropathy of the left hand were the symptoms. The peroneal muscles were paralyzed on the left side. Histological examination showed that the motor cells of the lumbar cord were very much diseased, while those of the cervical cord were normal. These changes were made out by the use of Nissl's method. The author looks upon the peripheral nerve lesions as secondary or dependent upon the central. Such an explanation may suffice for the minor tabetic amyotrophies, but it is entirely inadequate for the varieties considered here.

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[1] Read at the meeting of the American Neurological Association.

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[2] See article on "Morbid Anatomy and Pathogenesis of Tabes," Medical News, April, 1903.

[3] Revue Neurologique. 1896, No. 4.

The history of the first patient whose case I shall describe in detail is as follows:

Mr. M. was first seen by me December 28, 1895; he was then forty-seven years of age. His personal history previous to the beginning of his present illness was that he had married when twenty-five years of age and had been a widower since he was forty. He denied having had syphilis, but stated that all his life he had taken the risk of such infection. He admitted having had gonorrhea. In his business, that of a merchant, he had been a fairly hard worker, and during the last four or five years he had been worried because of business reverses. He was temperate in the use of spirits and an abstainer from tobacco. His family history was that his father and mother had died of apoplexy, the former at the age of sixty-five, the latter at the age of seventy-six. They had three children, one of whom died in infancy and the other in late childhood from acute diseases. So far as the patient knows there is no history of nervous disorder in the family. His own marriage had been childless.

Mr. M. enjoyed good health until about his forty-third year, when he suffered from an attack of pleurisy and pneumonia, and although he does not attribute his present trouble to that disease, he says that he has never really been well since he had it. A short time before the pleuro-pneumonia he was thrown from a horse and fell on his back. So far as he knows this fall did not injure him in any way, as he got up and rode on. After the attack of pleuro-pneumonia he suffered a great deal with his throat and had to go frequently to a physician, but I am unable to find just what the nature of the trouble was. Two or three months after the attack of pneumonia he noticed for the first time that both hands felt somewhat numb, and that there was some loss of strength in the left hand. At first this was not heeded particularly, as the numbness was variable in its intensity. A few months later he noticed that spaces were beginning to form between the bones of the left hand, and after this atrophy of the interossei had become quite marked, weakness and atrophy of the muscles of the right hand developed. He says that when the shrinkage began to show itself in the right hand the left hand got somewhat better, but it is probable that it got better merely by contrast. Later the atrophy extended to the forearms, arms and shoulders. All this time, he says, there was no pain, merely the sensation of numbness mentioned above.

Coincident with the development of the trouble with the hands, and possibly even before it, the patient remarked that it had become impossible for him to urinate in the customary way. At first the stream could be started with great effort after waiting patiently for a considerable time, but soon it became possible only to urinate when at stool, and he soon fell into the habit of urinating twice a day, at which time he would take a copious irrigation of the rectum. He had been constipated for some time before the beginning of his present trouble. He had begun the injections only after the difficulty with urination showed itself, and since the same time he had been wholly impotent, occasionally having a semi-erection in the morning when the bladder was over distended. About this time he noticed a sense of insecurity in walking in the dark, and soon fell into the way of having his room lighted at night so that he would be able to walk about more securely when he had to get up. About eighteen months after the symptoms showed themselves in the hand the right leg became lame so that he couldn't raise the thigh, or cross the right leg over the left without manual assistance, and the leg dragged so that he had to lift the knee very much higher on this side than the other in order to swing the toe free from the ground; in other words, foot drop on this side was an early symptom. All this time there was very little pain. The extremity wasted from the hip to the toes. Six months later a similar condition in the left lower extremity; the lameness was not quite so marked, but the wasting was just as conspicuous. It was particularly striking below the knee. During this time the wasting and paralysis of the upper extremities progressed somewhat, but with nothing like the progress that they did in the lower extremities. The condition of the bowels and bladder remained about the same. There was no incontinency of urine. The patient was able to get about with the aid of two sticks.

The patient had been under treatment by general practitioners and specialists from the first manifestation of his trouble, but treatment had not been of service. He had received electrical baths, hypodermic injections of strychnine, pilocarpine, the animal extracts, electrical treatment and massage. Three months before coming under my observation he began to complain of pain in the lower part of the spine, and of a sensation as if swarms of ants were crawling beneath the skin in the feet and legs. It might here be added that this symptom became very severe and added greatly to the burdens of the patient's life. He thought that the wasting and the lameness had apparently come to something like a standstill, as he seemed to be able to get about nearly as well as he had been any time during the six months previous. He complained of failing sight, of occasional severe attacks of dizziness, of involuntary twitching of the legs and drawing up of the knees. After being in bed a few hours the legs and thighs suddenly and forcibly flex, then extend, then the toes draw up and the feet twist about. These involuntary movements often occur every fifteen minutes, beginning at about 2 A. M. until he arises. They deprived him enormously of strength.

He does not complain of fibrillary twitching either when he is lying down or when he is quiet. The legs feel unwieldy and the feet very uncertain in their action. The only complaint of the upper extremities is that he cannot use the right hand very well. His business at this time compelled him to use a pen nearly all day, and the hand cramped so severely after a half hour or so that he would have to wait until the muscles got rested. Despite this he thinks that the hands are better now than they were six months ago. The left upper extremity is fairly strong and it does not give him any trouble.

On examination it is seen that the lower extremities and muscles about the pelvis, lower abdomen and right hand are the seat of marked wasting or atrophy. The remainder of the body seems fairly well nourished. The atrophy is most marked in the right hand, which has lost its normal contour, the thenar and hypothenar eminences being entirely flattened and the interosseous spaces hollowed. He is unable to adduct the fingers after they have been abducted, and to reflex the first phalanx of the thumb on the first metacarpal bone. The terminal phalanx of the thumb tends to remain in a position of forced extension. There is similar wasting, but to a much less degree in the left hand. The relative strength of the two hands is shown by the dynamometric registrations, that of the right hand being 20, that of the left hand 50. Although the upper extremities are thin, there is no real atrophy of any muscle or group of muscles. The direct myotatic irritability of the muscles of the upper extremities is lively, but the contractions produced by the percussion hammer are slow and vermiform. The wasting in the lower extremities is most advanced in the muscles supplied by the sciatic nerves, the right leg is affected more than the left. When he attempts to stand with the feet together the ankles tend to turn out and the knees to bend, and unless he is very careful he falls. The atrophy has begun to show itself in the shape of a pelvic girdle, i. e., extending around the upper part of the thighs and the lower part of the trunk. He experiences a great deal of difficulty in flexing and extending the thighs, but the adduction and abduction of the thighs is still performed very well. The most difficult movement of the feet for him to make is extension, especially of the right foot, which has assumed the position of foot-drop. On attempting to walk without shoes the right foot wobbles uncontrollably and is very apt to overthrow him. The electrical irritability of the muscles is characteristic of partial reaction of degeneration, i. e., there is less irritability to the faradic current than to the galvanic current; although both of them produce a response, that to the faradic is extremely sluggish and vermiform. The pupils are typical Argyll-Robertson, they are small, almost pin-point and wholly immobile to light and shadow, they contract in accommodation. There is no ataxia of the upper extremities. The sensory reflexes, plantar, cremasteric and abdominal are present and lively, save the lower abdominal, which is rather sluggish. Sensibility of the lower extremities is not acute, but still it was impossible to say that there was any real anesthesia or analgesia at this time. There was tenderness on deep-seated pressure over the sciatic and popliteal nerves.

The patient was put under treatment, which consisted of lenient massage, faradism to the muscles, galvanism to the spine, hypodermic injections of strychnine, and otherwise a general tonic plan of treatment. He grew worse gradually and notes of a complete examination made eight months afterward, viz., on the 1st of September, 1896, show that the patient walks laboriously with the aid of two sticks. The portions of the body in which the atrophy has made the most striking progress is in the lower pelvic girdle. The gluteus maximus muscles especially seem to have very nearly disappeared; there is very little power in this portion of the body. The movements of walking, swinging the legs, etc., are accomplished very largely by the aid of other muscles. The atrophy in the pelvic girdle is not quite so conspicuous on the anterior surface as it is on the posterior surface. The legs below the knees are in sharp contrast with the upper part of the thigh, insomuch as the atrophy here is relatively slight. The toes of the right foot are in a more or less constant state of flexor cramp, and he has very little voluntary power to move or wiggle them. Electrical examination of the abductors and extensors of the thighs shows that there is no response to the faradic current, while the anterior and internal crural muscles show a sluggish and vermiform response. The reaction to the galvanic current is diminished.

The immediate muscular irritability of the lower extremity is very sluggish, and no tendon response can be made out. The patient complains of paresthesia in the feet and along the outside of the thigh, and there is distinct analgesia over the peroneal nerves. Involuntary drawing up of the legs at night is quite as troublesome as it was.

The bowels and bladder do not act save on direct stimulation. It is necessary to inject large quantities of water into the rectum and colon before an evacuation can be secured. The bladder empties itself in part during defecation, but it is now necessary to use the catheter regularly. The atrophy of the muscles of the hand and right upper extremity has apparently ceased to progress. About this time the patient began to have disagreeable sensations of paresthesia and pain in the lower lumbar and sacral region.

The patient's condition grew gradually, very slowly but steadily worse. When he first came he was able to walk with a stick, a year later he was able barely to get about with two sticks, and needed great assistance in getting in and out of a car or carriage. A year later he could drag himself around upon crutches, and in another year he was in a wheel-chair. A note made April 12, 1898, states that the pain in his back is so severe that he cannot bear the weight of a truss which he has had to wear because of a right inguinal hernia. The left leg is now quite as much involved as the right. The toe-drop and foot-drop are so complete in each extremity that he stumbles and falls over everything. The bones of the lower extremities feel, he says, as if they had been beaten with heavy weights. The muscular twitching in both legs and in the right hand is extremely distressing, the fingers are much weaker, but the atrophy does not seem to have made any progress, it still confines itself to the thenar and hypothenar muscles of the first interosseous space. The formication on the bottom of the feet and the peroneal nerves, particularly the right, is intolerable. Vision has failed very much during the past year; now everything seems to be cloudy and misty. Examination of the optic nerve shows distinct optic atrophy, the pupils are the same as before, the bladder now can be emptied only by catheterization. The patient until this time had been of good courage, and faced his increasing infirmities bravely; he now became wofully despondent in the face of physical impotency and financial distress. He lasted about a year longer, during which time there was no particular change in his symptoms, the atrophy did not become more advanced than is indicated above, but he seemed to grow gradually weaker. A few months before his death he had some paroxysms of difficulty in breathing, but they were evidently symptomatic in character. The patient died rather suddenly, the symptoms immediately preceding death being those of pulmonary edema.

The spinal cord, pieces from many of the affected muscles and sections of the musculospiral, ulnar, sciatic and popliteal nerves were removed and prepared for microscopical examination.* The spinal cord was most profoundly affected in the lumbar region, and the morbid process diminished in intensity and extent upward from this region. Examination of sections taken from the first lumbar segment shows a degeneration of the posterior columns, and to a less extent of the posterior part of the lateral columns and the posterior roots. The bulk of the degeneration is borne by the middle third of the columns of Burdach and by the posterior roots. The lesion, which is a degeneration of the axis cylinders with a comparatively slight increase of glia tissue and very little vascular degeneration, is not, however, confined to that part of the column of Burdach to which the name "bandelettes externes" is given by the French writers. This part of the column is degenerated, but so also, particularly higher up, is the posterior part of the column of Burdach and the column of Lissauer. There is in addition some degeneration of the lateral part of the cord, especially of the posterior part of the cross-pyramidal tracts. The process does not seem to have involved all of the pyramidal tracts, nor is the intensity of the degeneration so great here as in the posterior columns. The involvement of this part of the cord is most evident in the upper lumbar region, least evident in the dorsal region, and slight in the cervical region. The uncrossed pyramidal tracts are not affected. There is no degeneration of other white columns of the cord. The anterior gray matter of the spinal cord in the lumbar region shows no abnormality, it seems to have preserved its contour and its normal constitution. The cell bodies of the peripheral motor neurones are of the customary size and shape and number, and accept the Nissl stain in the customary way. There are no vascular changes in the gray matter worthy of note. Many of the cells of the posterior columns are degenerated, atrophied and shrunken. The peripheral part of the posterior gray substance shows a greater amount of degeneration than the central part. There is little or no evidence of disease of the meninges.

Sections from the dorsal region show a different picture from that just described. The degeneration here is confined almost exclusively to the columns of Goll. The picture that is presented is almost identical with that of ascending degeneration in this column produced experimentally by section of posterior roots in the lumbar region. This degeneration of the columns of Goll can be traced upward into the cervical region, diminishing in extent, however, as we go upward, i. e., the degeneration occupies relatively a less area of these columns. In the lower part of the dorsal cord the columns of Burdach show slight degeneration, especially in that part of the column which borders on the posterior horn; in other words, the zone of the entering posterior roots. But as I have said, this area of degeneration soon disappears. The other parts of the cord are fairly intact. The lateral columns do not seem to be quite as normal in their appearance as they might be, but it is impossible to say that there is any degeneration of the axis cylinders here. The anterior gray matter is normal in its appearance and constitution, the columns of Clarke are well preserved, and the posterior gray matter presents no detectible change.

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*Reference to the mode of preparation is omitted. All the modern methods of staining the nervous tissues were used.

The state of the cord in the cervical region needs no extensive description. There is no lesion to be made out save the small area of degeneration in the columns of Goll corresponding to the degeneration spoken of below.

Examination of the spinal ganglia of the dorsal and lumbar region, stained with hematoxylin and, according to Nissl's method, did not reveal degeneration of the cell constituents of the ganglia, despite the fact that the degeneration of the posterior root fibers in the supra-ganglionary area was very evident. The anterior gray matter of this part of the cord was studied most carefully, especially sections stained by Nissl's method, but no alterations that are admitted to be truly pathological were found. Some of the cells showed a fair degree of chromatolysis, others irregular pigmentation, and here and there slight vacuolation. Dr. B. Onuf, who kindly looked through the specimens, subscribes to the statement that these cells could not be considered to be the seat of pathological alteration.

Sections of the ulnar, the median, the popliteal and other nerves show them to be the seat of alteration of the nature of parenchymatous and interstitial degeneration. The larger part of the nerve is transformed into fat tissue and fibrous interstitial tissue. Many of the nerve fibers that are not so transformed show partial degeneration. The fibers are swollen, twisted, here and there beset with fat globules. The walls of the blood vessels are thickened, particularly the external and middle coats, and the lumen is somewhat decreased. These thickened blood vessels are in striking contrast with the fat masses into which so much of the nerve has been transformed. The intensity and character of these alterations and changes in the nerve are more satisfactorily conveyed by the accompanying illustrations than by verbal description. In some of the nerves the process was more advanced than in others; for instance, in the ulnar and external popliteal it was most advanced. In the former more than one half of all the fibers are transformed into a homogenous fat mass, while sections from the sciatic nerve show that fibers having a more or less normal appearance predominate numerically, although the degeneration is here very striking. The sensory nerves seem to be involved only to a very slight degree. Sections from the radial and peroneal nerves show that the diameter of the nerve fibers is variable, and that the interstitial tissue is somewhat increased, but there is little or no alteration in the axis cylinders or in the sheaths of Schwann. The degeneration of the nerves is to be seen distinctly in many of the muscles. For instance, in the lumbricales. Here the irregular aspect and ragged appearance of the nerve fibers are very apparent, likewise the scarcity of them.

The lumbricales muscles fibers are themselves the seat of parenchymatous and interstitial alteration, but the process is neither so well marked nor so extensive as in the nerve. The muscle spindles are normal. This is well shown by Plate D, which shows two muscle spindles with thickened capsules. Between them a degenerated small nerve bundle cut obliquely is to be seen. Sections from the muscles of the thenar eminence, and from the gluteal muscles show most satisfactorily the change that went on in the muscles. It consists of a degeneration and atrophy of the muscle fibers, an increase of the interfascicular fibrous tissue, and a thickening of the blood vessels, but without proliferating endarteritis or anything suggestive of an obliterating process in the blood vessels. There is a very considerable fatty infiltration of the fibers, more in the sections from the gluteal muscles than from those of the hand.

The case therefore is one of tabes with muscular atrophy of neural origin, the lesions of the peripheral nerves being a degenerative neuritis. The lesions of the muscles are very similar to those of neuropathic atrophy, a parenchymatous decay with accompanying fatty infiltration and slight connective tissue increase. The clinical features of the tabes were not typical, neither were the lesions of the cord. In my study of the profounder forms of tabetic amyotrophies, I have been very much struck by a statement made by Chretien and Thomas[4] to the effect that there is a form of tabes that begins with muscular atrophy in which tabes symptoms remain very much in the background. In none of the three cases that I am reporting in this article was the existence of tabes suspected until it was revealed by the physical examination. Strümpell, in commenting upon the cases of the above mentioned writers, remarks that paralyzed legs do not show ataxia. But that has nothing to do with the subject. The point is, that in these cases, such as I report, there are no subjective symptoms of tabes of any moment, and the disease tabes does not comport or display

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[4] Revue de Médecine, 1898, p. 886.

itself in the ordinary way. The best proof of this is that tabes is only revealed by the examination. It may be that the atrophy is such a dominant symptom that it obscures the tabetic symptoms. But the atrophy does not always come first in these cases. For instance, in a case somewhat similar to mine reported by Goldscheider,[5] the following symptoms developed first: Girdle-feeling, diplopia, right-sided ptosis, numbness in the legs. At the time of examination there were present: Right-sided ptosis and mydriasis, ataxia of the lower extremities with profound muscular atrophy, slight ataxia of the hand, sensory disturbances in the lower extremities, and loss of knee-jerks. In the course of the disease the atrophy became more and more pronounced, and the picture of tabes quite typical. The patient was a woman forty years old who denied syphilis and alcoholism. On microscopical examination there was found in the middle of the cervical region degeneration of Goll's column, and likewise a similar area which extended into the anterior portion of Burdach's column, reaching the lateral border between the middle and posterior third of this. This area was separated from Goll's column and from the posterior horn by an area equally wide of intact white matter. The degeneration increased as the examination was carried lower and lower, so that in the dorsal region nearly all of the posterior columns was involved by the degenerative process. About six cm. above the beginning of the lumbar cord there was a limited degeneration of the lateral columns especially at the periphery. This area was larger in the lumbar region and two cm. below the beginning of the lumbar segments it had the appearance of a wedge driven into the lateral part of the cord. In a number of peripheral and sensory motor nerves there was found intense atrophic degeneration. Other cases of tabetic amyotrophy in which more or less involvement of the lateral part of the cord has been found, are reported by Schultze and Strümpell.

In this JOURNAL of February, 1894, I reported a remarkable case of progressive muscular atrophy with locomotor ataxia, which it may be said in passing has been referred to by some European writers as one of probable syringomyelia. If one is willing to make the diagnosis of the latter disease when the symptoms of tabes and progressive muscular atrophy co-exist, I cannot gainsay him the privilege. I had the case under observation for many years and saw him last three years after the report of the case was published. During this time no features diagnostic of syringomyelia were present.

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[5] Zeitschrift f. klin. Med., Vol. xix.

The patient, a man fifty years old, had been a hard drinker in his early life, and had contracted syphilis when twenty-five years old. When he was thirty-six years old, and at a time when he was drinking freely of alcoholic liquors, he noticed for the first time that the arms and the legs were getting weak, and that he had occasional attacks of pain in the legs. Two years later he lost control of the bladder, and was so weak that he had to go to a hospital. While he was in the hospital his upper extremities became quite powerless, the hands and shoulders began to get smaller, and continued to do so until they reached the condition in which I found them, absolute powerlessness and flaccidity of the upper extremities. Soon after he went into the hospital he suffered from incontinence of feces as well as of urine. After two years in the hospital, he had regained control of the sphincters, and his legs became stronger. It was at this time that he began to complain that he saw double, and of lightning pains in the lower extremities. These pains were so severe that for weeks at a time he could not sleep. Sexual power was lost rapidly, and he had occasional incontinence of urine. From this time he was unable to walk in the dark and had difficulty in descending steps. He says he feared that he would fall as he could not grasp the bannister with the hands. The wasting of the upper extremities, which had been progressing during this time, began first on the right side, and has always been more marked there, although the left is extremely affected. The wasting involved the flexors and extensors equally and no suspicion of the "claw hand" ever developed. The wasting at first was rapid, but after three to four years it progressed very slowly.

Examination made in April, 1893, showed: On inspection, the patient when at his ease assumes a posture with the head bent forward so that the chin nearly touches the chest. (Photographs of the patient are to be seen in the original article.) When asked to hold the head erect the chin is tilted upward and the head is inclined a little backward. The former is his habitual posture. The upper extremities hang like flails from the shoulders. They are completely powerless. By swaying the body he is able to start them in a swinging motion. The hands look as if they had been flattened with a rolling pin; the eminences and depressions have entirely disappeared. The forearms and arms have lost almost completely their muscular tissue, some fat still remains. The head of the humerus on the right side has dropped from its socket so that a depression the width of a finger is left. On the left this is also apparent, but to a less degree. The muscles of the shoulders and back presenting the most extreme atrophy are, on the right side, the deltoid, supraspinatus, and infraspinatus, serratus anticus (which presents a remarkable degree of wasting), trapezius, with the exception of its occipital bundles, teres major and minor. The latissimus, pectoralis major and sternocleidomastoid are affected to a less degree. The rhomboids are quite well preserved when contrasted with the right.

There is no fibrillary tremor in any of the partially wasted muscles and no response to tapping. The size of the lower extremities is not far from normal, but there is a suspicion of atrophy in the left peroneal group.

There is a marked Romberg symptom, and inability to stand on one foot or to raise himself on tiptoes, and inability to walk backward. There is considerable ataxia of locomotion. The knee-jerks and ankle-jerks cannot be elicited. In the lower extremities there is marked diminution of sensibility, especially for pain, and sensation is greatly delayed. He says "now" in response to a severe prick of a pin in from three to four seconds in different parts of the lower extremities, and the maximum pain is not attained for several seconds. Sensibility of the upper extremities is well preserved. Temperature sensibility normal. The pupils react moderately to accommodation, but not to light. The patient complains of great heaviness of the lower extremities, paroxysmal lancinating pains, and of girdle sensation. Cold weather or exposure aggravates his symptoms markedly and causes frequently distressing weakness of the sphincters. The atrophy of the muscles has apparently come to a standstill.

The most legitimate interpretation to put upon this case, it seems to me, is that the patient had multiple neuritis probably of alcoholic origin, and that the motor nerves of the upper extremities and shoulders were so profoundly diseased that they never regenerated. Whereas, those of the lower extremities being very much less affected, quickly recovered. The most serious objection to this interpretation is that the sensory nerves of the upper extremity were spared. It may well be, however, that at the onset of the probable neuritic process they were affected as well. Despite the fact that the toxic effect of alcohol is most apt to be expended upon the sensory nerves, cases of alcoholic motor neuritis are not uncommon. If we do not adopt this idea of the muscular atrophy the case must be looked upon as one of muscular atrophy of spinal origin, and against this conception a number of factors speak. In the first place the suddenness of onset, rapidity with which it developed and destroyed the muscles, the uniform manner in which it affected all the muscles and its absolute cessation are very unlike the manner in which spinal progressive muscular atrophy comports itself. The most distinguishing clinical feature of the latter disease is the selective manner in which it destroys groups of muscles of an extremity, leaving others untouched. The insidious way in which it attacks the muscles, the existence of fibrillary twitchings, the course of the disease are all very different. The lesion may have been a chronic anterior poliomyelitis, in fact there is nearly as much to be said in favor of this diagnosis as of that of motor neuritis.

The third case of muscular atrophy associated with tabes that I shall speak of is one that is mentioned here more because of the interesting question in diagnosis that it presented rather than because it comes in the category of the cases that I have been describing: D. T., a barman, forty-two years old, contracted syphilis at the age of twenty-four, and had very little specific treatment. A year later he had an attack of iritis. When thirty-four years old he had a cerebral seizure in which he was very dizzy and fell to the ground but was not unconscious. For two days he did not speak correctly. There was no paralysis accompanying or following the attack. Before this attack he had suffered for a number of months with headache. Four years later he had a similar attack. His present illness was preceded for a number of weeks by mental depression. On August 18, 1902, while eating supper, he fell from his chair to the floor. He was only partly unconscious he says, for he knew those that helped him to bed. The following day the right arm and leg were equally paralyzed and he had dysarthria, but no real aphasia. When he came to the hospital the hemiplegia had so far recovered that he was able to walk slowly with the aid of a stick. The degree of recovery was greater in the leg than in the arm. On examination of the right upper extremity a marked wasting of the muscles of the shoulder cap was seen. This was so great that the head of the humerus dropped from the glenoid cavity. There is no evidence of fibrillary contraction in the atrophied muscles, and they react sluggishly to the electrical currents, more so to the faradic than to the galvanic current. Further examination shows absent knee-jerks and ankle-jerks, pronounced hypotonia of the left lower and upper extremities, Romberg's sign, Argyll-Robertson pupils, diminished sensory acuteness over the circumference of the body, corresponding to the 9th and 12th ribs, and in the feet and legs. Plantar jerks exaggerated, both of the extensor type, the right constituting a typical Babinski phenomenon. The patient has scarcely any symptoms of tabes save impotency and occasional incontinence of urine, and the disease was not suspected until its existence was revealed by the examination.

The muscular atrophy which he shows may be explained in one of two ways, either it is dependent upon a peripheral lesion, such as a neuritis of the circumflex and other nerves of the brachial plexus, or it is due to a thrombosis of the cornual branch of the anterior spinal artery, similar to the thrombosis within his brain that caused the hemiplegia, with resulting limited neurone destruction. At the present time it is impossible to say upon what the atrophy is dependent. The fact that it seems to be slowly progressive speaks in favor of the latter view. The case is of interest in showing that an attack of cerebral apoplexy with consecutive degeneration of the crossed pyramidal tracts, as evidenced by the Babinski phenomenon, does not cause a return of the knee-jerk. In this case it is extremely probable that unequivocal signs of tabes antedated the attack of cerebral thrombosis upon which the hemiplegia is dependent.

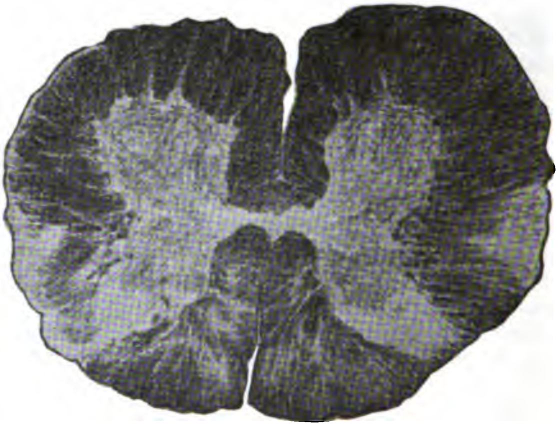

FIG. 1

Some of the cases of tabes and muscular atrophy that have recently been published in addition to those already mentioned, are the following:

Wagner[6] reports two cases. The first case, a thirty-six year old laborer, who had been ill eighteen months, said that weakness and wasting of the extensors of the right foot set in shortly after a course of mercurial inunctions. The affected muscles showed partial reaction of degeneration. The author thinks that the neuritis may have been of mercurial origin, but considering that the reality of mercurial neuritis is not generally granted, this seems very improbable.

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[6] Inaug. Dissertation, Berlin, 1896.

The second case, a woman forty-seven years old, with signs of lues and symptoms of tabes, complained of weakness of both forearms; the thenar and hypothenar eminences were flattened, the interossei likewise atrophied, giving the typical claw-hand. There was partial reaction of degeneration in the nerves.

V. Ufen[7] reports a case of tabes dorsalis and muscular atrophy, in which there was wasting of the muscles of the hand, shoulder and the thighs. There were no electrical changes. At the anatomical examination there was found a well marked spinal meningitis in addition to the typical posterior cord root degeneration, but no atrophy of the anterior horns. The peripheral nerves were not examined, but Ufen does not doubt the neuritic nature of the muscular atrophy, as the anterior horns were normal.

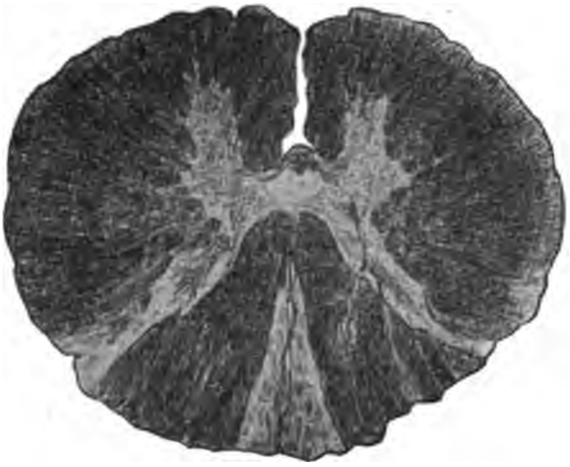

FIG. 2

Jolly[8] has recorded a case of tabes with muscular atrophy which was quite profound in one lower extremity and in both upper extremities. There was very little disturbance of sensibility in the arms.

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[7] Inaug. Dissertation, 1896, Kiel.

[8] Berliner Gesellschaft f. Psychiatrie u. Nervenkrankheiten, March 9, 1891.

Chretien and Thomas[9]. Their first case in which there was atrophy of the lower extremities of rapid development and slight atrophy of the arms, showed on autopsy in addition to tabes, widespread degeneration of the ganglion cells of the anterior horns in the lumbar region. The changes in the peripheral nerves were distinct, but not so marked. In their second case the anterior horns showed only slight changes, whereas there was profound peripheral neuritis.

E. W. Taylor[10] has recently published a very striking case of progressive muscular atrophy of the muscles of the hands and forearms, which, from the clinical point of view, seemed to indicate degeneration of the peripheral motor neurones.

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[9] Revue de Mèdecine, 1898, November, p. 886.

[10] Boston Medical and Surgical Journal, 1902.

## EXPLANATION OF PLATES.

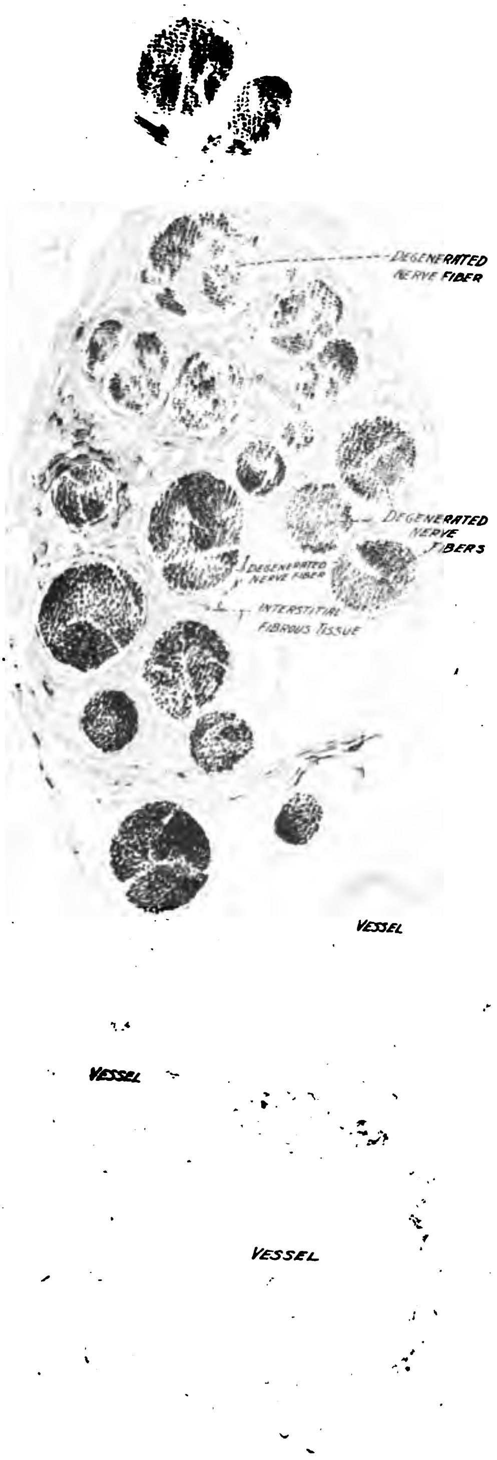
Plate A—Transection of the ulnar nerve showing the greater part of the nerve transformed into fat tissue and interstitial fibrous tissue. Also the enormously thickened blood vessels, take up a considerable portion of the tissue that remains. Of the still remaining nerve bundles many show considerable degeneration.

Plate B—Showing four degenerated nerve bundles, A, B, C, D, in a lumbricalis muscle.
Stained after Pal counterstained with Picro-acid-Fuchsine.
The nerve fibers show accordingly as black stripes in the longitudinally cut bundles B, C, D. Note the ragged appearance and scarcity of nerve fibers in the four degenerated bundles A, B, C, D, especially in A and B.
Fat=fat tissue.

Plate C—From the same section as Fig. B. showing, also in a lumbricalis muscle, numerous degenerated nerve bundles in which the scarcity and irregular ragged aspect of the nerve fibers are very evident.
Verm.=vermiform muscular fibers.
Fat=fat tissue.
Atr. M.=atrophic muscular fibers.
M. transv.=transversely cut muscular tissue.

Plate D—Taken from a lumbricalis muscle from the same section as plate B and C. It shows two muscle spindles, with thickened capsula, but apparently well preserved muscle fibers. Between the two muscle spindles lies a degenerated little nerve bundle, cut obliquely.
M and M are two muscle fibers of the lumbricalis muscle.

PLATE A.


DEGENERATED NERVE FIBER
DEGENERATED NERVE FIBERS
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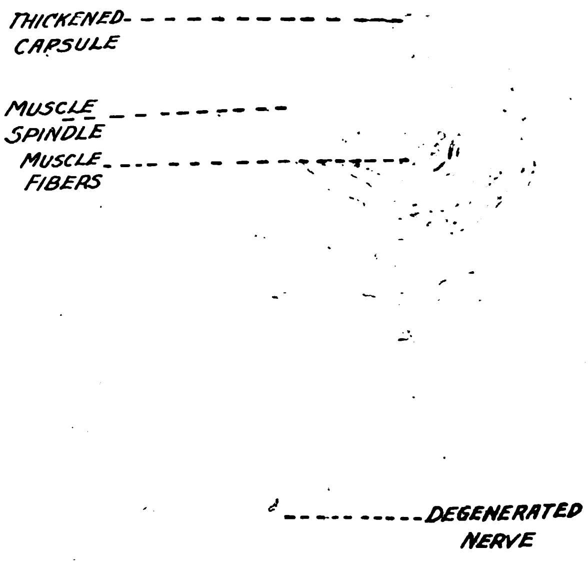
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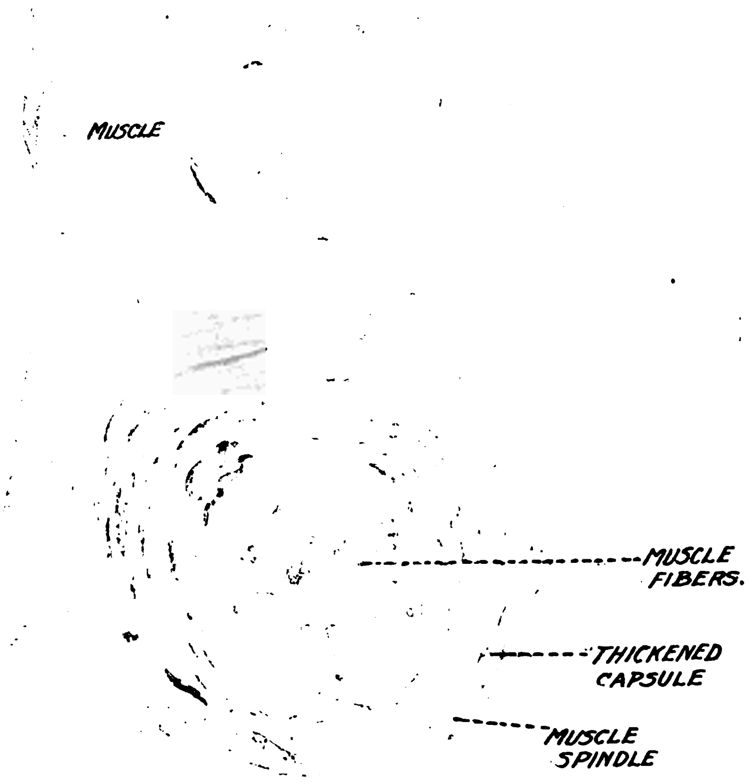
MUSCLE
MUSCLE FIBERS.
THICKENED CAPSULE
MUSCLE SPINDLE

# CONTRIBUTION TO THE STUDY OF THE ACHILLES-JERK AND THE FRONT-TAP.[1]

By G. L. Walton and W. E. Paul,
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The recent activity in the study of the reflex mechanism has brought to light a great number of movements involving a large part of the musculature. The importance of each new reflex is urged, the character of each is minutely discussed, and the priority of observation disputed. It would not be right at present to deny the importance of any of them, for we can all remember the time when we held, and taught, that the plantar reflex with its variations was of no import practically, and was never likely to take a place among the major tests in diagnosis. This reflex now stands perhaps at the head of the list from which it was formerly excluded. Whatever prominence these new reflexes may attain in the future, two phenomena, though long recognized, seem not yet to have received quite the attention they deserve, namely, the Achilles-jerk and the front-tap. The Achilles-jerk has been extensively studied, but has not taken the place it should, at least on this side of the water, as a routine test of equal importance with the knee-jerk; the front-tap has not even been extensively studied.

The object of this paper is to emphasize the importance of the one and to contribute toward the study of the other.

## THE ACHILLES-JERK.

The result of our study of this (so-called) reflex coincides with those of most observers. We have found it in health equally constant with the knee-jerk as, with the exception of Strasburger, have other observers. (Bramwell, Sarbo, Cestan, Schoenborn, Kollarits.) We have also found it more uniform in excursion and more easily elicited than the knee-jerk. We have found it disappearing early in tabes, and regard its loss quite as typical of this disease, as the absence of knee-jerk. These results are in accord with those of other observers (Babinski, Mills, Collins, Bramwell, Seyer, Goldflam, Trumporszki). We have found it increased as a rule in hypertonic, and decreased in hypotonic conditions.

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[1] Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

We have employed the various methods suggested for testing this reflex, and find little to chose between having the patient kneel in a chair, and having him sit in a chair with the knee bent to a right angle, or to an angle slightly more acute. If the patient is in the latter position the heel can be seen to rise if the reflex is active, but it is best to place the hand on the calf and to judge its activity by the muscular contraction rather than by the movement of the foot. For purposes of demonstration the kneeling posture is preferable. It is better to have the patient kneel on a cushion or a shawl rather than on a hard wood seat, for many persons find the latter so uncomfortable as instinctively to put the muscles under a tension which interferes with eliciting the reflex. Some find it difficult to relax in this situation in any event, and for such the sitting, or even, as Sarbo suggests, the recumbent posture may be tried to advantage. Muscular tension on the part of the patient interferes less, however, with the Achilles than with the knee-jerk, an additional argument in favor of its routine use. For patients in bed we use the methods of Strasburger and Ziehen (with knee straight and flexed respectively), with such modifications as the circumstances demand.

In the majority of cases the reflex can be obtained without removing the shoes, but this precaution should never be neglected in doubtful cases. In some instances it is best obtained by tapping the middle of the tendon, say two inches above the os calcis, in others by tapping the tendon close to its insertion, and sometimes better on one side. Less technical skill and less practice are required to obtain this reflex than to obtain the knee-jerk. It follows that as this test comes into general use, the records of those not specially skilled will be more reliable than they are at present regarding the knee-jerk.

*The Achilles-jerk in health.* We have examined 500 persons of both sexes varying in age from five to eighty-two years, either in apparently perfect health or suffering from some trouble independent of the nervous system, and have failed to elicit this reflex on both sides in only one instance, on one side in four. In these five instances the knee-jerk was present. The knee-jerk was present in every case; in two it required reinforcement on one side, but was easily elicited on the other, and in eleven the knee-jerk was so feeble as to require repeated tests with reinforcement; in these thirteen instances the Achilles-jerk was easily demonstrated. In a single case, an active knee-jerk was combined with an Achilles-jerk so feeble as to be elicited with great difficulty.

One of the cases of diminution of patellar and presence of Achilles-reflex was a man of eighty-two; in another, a man of forty-nine, diphtheritic paralysis had occurred at the age of twenty-five, before which the knee-jerk was present and active. The latter history led to an inquiry as to the previous history of the cases of unilateral feebleness of knee-jerk. It appeared that one of these individuals, a healthy man of twenty-eight, had in infancy a severe attack of diphtheria not followed by paralysis; the other gave a history of repeated and severe sore throats.

The question seems pertinent then, *whether prior toxic influence may not sometimes cause the enfeebled, or even absent, knee-jerk occasionally found in health.* If this was the case in the two instances cited, these observations only serve further to illustrate the hardiness of the Achilles-jerk, since this phenomenon was active in both.[2]

Though the excursion and activity of the Achilles-jerk in our cases varied to a certain extent in different individuals, it was constantly noted that these variations were appreciably less than those of the knee-jerk. In an occasional case it has been somewhat stronger on one side, as has the knee-jerk.

The result of these observations, coupled with those of others, led to the conclusion that the routine test of the Achilles is of equal importance with that of the patellar reflex in eliminative diagnosis and in the establishment of neural health.

*The Achilles-jerk in disease.* In thirty-five cases of tabes dorsalis we have found none with preservation of this reflex except one case in which the symptoms were unilateral and all reflexes were normal on one side. We have found two cases with present knee-jerk on both sides and with absent Achilles-jerk on both sides.

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[2] It is worthy of note in this connection that Pfaundler found the knee-jerks absent in 27.5 per cent of 200 cases of croupous pneumonia in children.

Dr. McCollom, who, as the head of the contagious service at the Boston City Hospital, has seen some 10,000 cases of diphtheria, states in answer to an inquiry that the knee-jerk is wanting in the majority of serious cases of this disease. He questions whether cases of so-called croupous pneumonia are not frequently cases of diphtheria of the lungs, the physical signs of the two affections being identical.

These observations tend to corroborate the opinion of most other observers, that the Achilles-jerk is, as a rule, the earlier to disappear in tabes.[3] This is a most important practical point in diagnosis. Suppose, for example, we are consulted by a person with feeble or absent knee-jerk. If we find his Achilles-jerk present we may reassure him with considerable confidence as to any suggestion of tabes in his absent knee-jerk. If the Achilles-jerk is absent we cannot so reassure him, though it would be going too far to say that this combination establishes tabes, for it may also be found in other hypotonic conditions.

In other directions our data while too few to be used alone for statistical purposes, may be suggestive. We have found this reflex present, generally active, in thirty-eight cases of epilepsy usually accompanied by active knee-jerk; it was diminished in only one case, a case in which the knee-jerk was present. All these patients were taking bromides, generally not less than 60 grains daily.

This finding is in accord with the statement of Clark, who finds the knee-jerk generally active in this disease except during the attack, when it disappears. He regards this activity as an exhaustion phenomenon. As far as we have been able to discover, no other statement has been made regarding the reflexes in this disease, excepting during and immediately following the attack. In exophthalmic goiter we have found the Achilles-reflex present, generally active, in eight cases, wanting in none. In neurasthenia, hysteria and other neuroses we have found this reflex always present, but less active and disagreeable than the knee-jerk. In fourteen cases of paresis both Achilles-jerks and knee-jerks were increased in seven, decreased in two, average in five. The knee-jerk and Achilles-jerk were exaggerated in twenty-four cases of hypertonic paralysis including hemiplegia, wanting in three old cases of hemiplegia with contracture. In hypotonic conditions the Achilles-jerk has always been absent. The Achilles-jerk was wanting on the side of sciatic pain in one case of osteoarthritis of the spine.

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[3] Bramwell's results varied slightly from those of other observers in that although he found loss of both reflexes in 48 cases out of 52 examined, in the other four the loss was equally divided between the two reflexes.

### THE FRONT-TAP.

The study of this reflex (named by Gowers) seems to have been meager. Search through the literature reveals the fact that neither its physiology nor its diagnostic significance has received attention. The method of testing the front-tap consists in smartly tapping the tibialis anticus with a Taylor rubber hammer (less advantageously by the finger), the examiner meantime flexing the foot dorsally, and holding it in this position, the ankle resting on the knee of the examiner or on the edge of a stool (preferably slightly lower than the chair upon which the patient is seated, or with the leg extended if the patient is in bed). The reflex consists in plantar flexion of the foot.

Gowers suggests that the stimulus may pass through to the flexors of the calf, and he has called attention to the fact that the vibration may be felt in the gastrocnemius. This explanation is weakened by his observation that the phenomenon is less readily produced, if at all, by tapping the shin-bone. As regards the significance of the front-tap, Gowers speaks of it as occurring *when the reflex phenomena are in excess,* and again as a very delicate test of increased irritability.

The explanation offered by Gowers regarding its production does not seem quite satisfactory, and while we are not sure that we can better it, we would suggest that it may be allied in a way to the ankle-clonus, though not of its sinister import. May it not be the case that in taking the front-tap the position of the foot in extreme dorsal flexion (and this generally is the most favorable position) puts the tendo-Achillis under such tension that the trifling tendency for the tibialis anticus to contract and thus still further dorsally flex the foot is enough to stimulate, or tend to stimulate, the gastrocnemius into a contraction? An observation occasionally made tends to confirm this view. Namely, in a rare instance evident dorsal flexion of the foot precedes plantar flexion. In fact, sometimes the only movement is an upward one. In many, perhaps in most, cases in which the front-tap phenomenon is present, there is visible contraction of the tibialis anticus.

Whatever the mechanism of this phenomenon may be, its significance seems to have been not completely interpreted, probably because no systematic test has been made of its presence or absence in health.

*The front-tap in health.* We have made this test in 500 persons, all of whom were either in apparent health or were subjects of disorders having no recognized influence on the central nervous system. Of these individuals 200 were males and 300 females. The ages varied from six to eighty-two.

The result of this examination showed the front-tap present in 75 males, or 37.5 per cent, and in 124 females, or 41.3 per cent. In many of these cases it was extremely lively. In an occasional instance the difference between the two sides was sufficiently marked to cause the phenomenon to seem absent on one side at the first test while present on the other, but in such cases more searching examination generally established its presence on the side first tested.

It is possible that in a larger number of these cases the reflex could have been teased out by persistent effort, but whether this is so or not the fact seems established that the *mere presence of the front-tap, even in active form, does not militate against perfect health.*

In order that the test might be made in one series from which the question of ill health could be practically eliminated, 59 picked young women taking the course in the Boston Normal School of Gymnastics were tested through the kindness of the director, Miss Amy Morris Homans.

Out of the 59 cases the front-tap was present in 15, that is, in about 25 per cent.

This series is mentioned as demonstrating the presence of the front-tap in health. The number of cases is too few to warrant drawing a comparison between the athletic and the average individual in respect to the front-tap, but further elaboration of this comparison in the matter of all reflexes might prove of interest.

We have not found the position of the knee, straight or flexed, of importance in eliciting the front-tap. One precaution should be mentioned, however, in this connection, namely, to avoid pain in patients with sciatica, osteoarthritis of the spine, and allied conditions, it is well to have the foot much lower than the knee, or else to make the test with the patient in the recumbent posture. In very rare instances healthy persons complain of slight discomfort if the leg is placed as pictured by Gowers, a fact which has led us to allow the knee of the person examined to be slightly flexed.

*The front-tap in disease.* Our observations tend to show that this test, though of less positive value in diagnosis than the Achilles-jerk, may be of material aid in connection with other signs and symptoms.

In neurasthenia, hysteria and other neuroses we have found the proportion of front-taps greater than in health, namely in 59 out of 80 cases (70.4 per cent); namely, hysteria, present in 14, absent in 6; neurasthenia, present in 31, absent in 9; other neuroses, present in 14, absent in 6. In 8 cases of goiter it was present in 7 (87.5 per cent). In an epileptic attack the front-tap disappears with the knee-jerk. In 100 cases of epilepsy the front-tap was present in 75 (in several only on one side), absent in 20, impossible to test on account of position of limbs and mental condition in 5.

We have in no case found the front-tap present in tabes or other hypotonic affections; in paresis we have found it present in 8 out of 14 cases. In other diseases involving hypertonic conditions, including hemiplegia, we have found it present, generally exaggerated, in 24 out of 28 cases. Three of the cases in which it was wanting were old hemiplegics with contracture and absence of other reflexes; one was a case of probable meningitis and encephalitis of infectious origin with absent knee-jerk and present ankle-clonus, a combination elucidated by Mills, the absent knee-jerk being due, perhaps, in this case, as in that of Mills, to neuromuscular infection, though autopsy could not be obtained to decide this question.

For opportunity to add to our statistics we are indebted to our colleagues in the Massachusetts General Hospital, to the staff of the Eye and Ear Infirmary, the State Hospital at Tewksbury, and the Long Island Hospital, also to Dr. Fuller of the Adams Nervine Asylum, and Dr. French of the State Hospital at Medfield.

From the numerous *instances illustrating the practical aid in diagnosis furnished by these reflexes* we will cite two only, both of general paralysis, one showing the hypertonic, the other the hypotonic variation in this disease.

Case I. A man forty-four years of age complained of numbness in the left side and of frequent epileptiform attacks of recent onset. Both the patient and his wife seemed inclined to attribute his condition, largely at least, to a blow received over the right ear from an iron bar six months ago. There was brief unconsciousness, the patient being able to get up and go home. There were evidences pointing to exaggeration, for example, the extreme tenderness complained of at the point of injury seemed to disappear when his attention was distracted; the test for anesthesia also was inconclusive.

The knee-jerks and pupils were normal and there were no other definite signs elicited by physical examination until the *Achilles reflex* was tested; this was *absent on both sides.* Further examination of the mental condition showed inability to carry test sentences in mind or to repeat them correctly. The writing also showed evidence of mental deterioration. In keeping was the statement of his wife that there was marked change in disposition and that he was not to be depended upon for errands or other duties.

In this case the absence of Achilles reflex was the one sign which enabled us to diagnosticate organic disease (general paralysis) with a confidence which would hardly have been justified under the circumstances either from his mental exhibit or from the history.

Case II. A man of forty-four years of age, for some time under observation at the Massachusetts General Hospital, had been regarded as a case of early tabes notwithstanding the preservation of knee-jerks and Achilles reflex. The diagnosis was based on the characteristic pupils and other symptoms.

The routine test revealed an *active front-tap.* This led to a reconsideration of the diagnosis, and further examination demonstrated the mental condition characterizing the early stages of general paralysis.

## CONCLUSIONS.

1. The Achilles-jerk is practically as constant in health as the knee-jerk. This reflex varies less in health than the knee-jerk in excursion and activity, and is the most easily elicited and uniform of all tendon reflexes.

2. The Achilles-jerk disappears, as a rule, early in tabes dorsalis, and its absence is as diagnostic of that disease as is loss of the knee-jerk. We have not seen a case far enough advanced to establish tabes with persistence of the Achilles-jerk, except one case in which both the knee-jerk and the Achilles-jerk were present on one side only.[8] We have observed bilateral preservation of knee-jerk and loss of Achilles-jerk in two out of five cases of tabes.

3. Enfeeblement of knee-jerk in health on one side or both

may be due to prior toxic influence, as diphtheria. This may also be true of the Achilles-jerk, though in the one case in which it could be demonstrated of the knee-jerk, the Achilles-jerk was normal. Further observations on this point are desirable.

4. The front-tap is present (generally on both sides) in about 40 per cent of individuals in ordinary health; in some it is very active. It follows that its presence alone, even if active, does not establish disease, nor indicate excessive irritability of the nervous system.

5. In organic disease the front-tap is generally increased with the other reflexes in hypertonic, and decreased (generally wanting) in hypotonic states.

6. In the so-called functional disorders, hysteria, neurasthenia, and unclassified psychoses, we have found the front-tap present in 71 per cent of cases. In epilepsy we have found it present in 75 per cent of cases. The test may therefore here prove of aid in combination with other findings, though its mere presence or even activity is not of positive diagnostic value, nor does its absence negative the existence of neuropathic conditions.

7. Both these reflexes deserve to be placed upon the list of routine tests for purposes of diagnosis. This is particularly true of the Achilles reflex, which is of the greater positive diagnostic value.

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# THREE CASES OF PROGRESSIVE MUSCULAR DYSTROPHY OCCURRING IN THE MALE MEMBERS OF A SINGLE FAMILY, AND COMMENCING AT THE SAME AGE IN EACH.

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In the following report I desire to put on record three cases of progressive muscular dystrophy occurring in a single family, two of which were seen by the kindness of Dr. Edward Evans, of La Crosse, Wis., at whose suggestion they are published. The cases seem noteworthy in the following points:

(1) The apparent absence of etiological factors and especially of hereditary influence.

(2) The susceptibility of the boys and immunity of the girls of the family, to the disease.

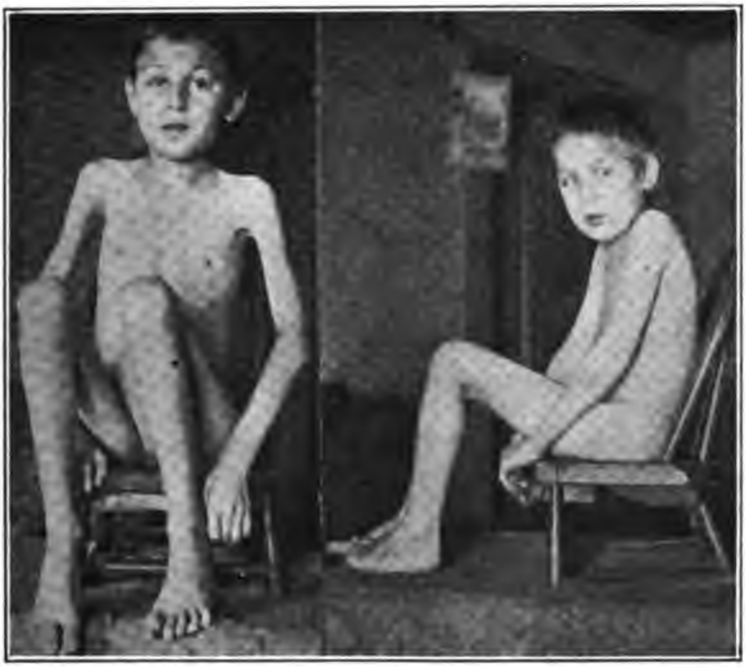
(3) The remarkable uniformity of the cases in age of onset and in the course of the disease.

The patients are the children of Christian W., born in Germany in 1847, and Marie, his wife, born in Germany in 1857, who were married in 1875, and are the parents of ten children. The parents emigrated to the United States in 1873. The father has been employed as a day laborer, and at present works in a railroad coal yard. He has always been well except for "rheumatic attacks," which affect his back. Of these he has had three. He has not a marked alcoholic history, but is a moderate drinker of beer. The mother has always been well except for measles and a severe attack of scarlet fever when she was twelve years of age. She was then ill for twelve weeks. No history suggestive of syphilis is to be obtained in case of either parent. The father and the mother both have brothers and sisters, all of whom are reported as well. No history could be obtained of paralysis or muscular trouble of any sort in the family of either parent.

The children are: Charles, born in 1876, death from burns in 1879; Ida, born in 1879; Bertha, born in 1881; Hugo, born in 1883, death in 1898; Allie, born in 1886; Edward, born in 1889, death in 1902; Fritz, born in 1891; Meta, born in 1895; Lillian, born in 1896; Esther, born in 1899.

The births were all natural and not difficult. The girls of the family are all in good health. They are as yet unmarried. The boys are affected with progressive muscular dystrophy of a scapulo-humero-femoral type. The histories of the boys as given by the mother are very similar and are as follows:

Hugo, the oldest, was always pale and sickly, and complained much of his stomach hurting him. When about five years of age he seemed to be getting weak; his knees would give way and he would fall. He would have to help himself up by holding on to a chair or convenient object. He was never subject to convulsions or febrile attacks. When about nine years old he became unable to walk at all. He sat in a chair for six years, when in the mother's words "he began to cough and coughed for six weeks and died."



Figs. 1 and 2—Photographs of two of the patients with progressive muscular dystrophy. Edward (the larger boy) and Fritz (the smaller boy).

Edward, when a year and a half old, passed "blood" in his urine for a week. He has never done so since, and has never been really ill since. He played about like other children until he was five years old, when he began to get pale and weak and lost his appetite. He was thought by some to have rickets, by others worms. The weakness continued, and then his knees began to give way. In his sixth year he complained of pain in his elbows, knees and ankles (at present, 1900, he has no pain anywhere). The boy kept around but grew weaker and slowly wasted. The wasting, however, was not extreme until 1897. During the next year the patient became so weak he could not walk, and since then he has sat in a chair continually.

Fritz, the youngest boy, was never ill until his fifth year, when he began to get weak as had his two brothers before him. This weakness continued until in the early part of the year 1900 he found that he could not walk, and took to a chair. In no one of the boys could a history of any hypertrophy of the muscles previous to the atrophy be obtained.

Edward and Fritz were first seen in March, 1900. Edward (Fig. 1), aged eleven years, showed extreme pallor, a mask-like expressionless face, and very marked muscular wasting. He was seated in a low chair with his legs flexed on his body and at the knees, and his feet held in an equino-varus position. His chest was flat and his abdomen slightly protuberant. His pupils were equal. Consensual and direct reflexes present. Patient raises eyebrows, smiles, shows teeth, etc., to normal extent, and apparently with normal power. He can whistle well. Degeneration of muscles of shoulder girdle is extreme, pectorals almost indiscoverable, trapezius, deltoid, infraspinatus extremely wasted, supraspinatus less so. On attempting to lift patient with hands placed beneath his arms, his shoulders slide up almost to his ears. Wasting of the upper arm muscles is also marked. The biceps is contracted and like a tense cord resists full extension of the arm at elbow. Muscles of forearm and hand show no atrophy. The grip is good. Patient is able to raise his right arm over his head and can hold it at the horizontal; cannot raise left arm to the horizontal.

The atrophy in the legs is confined chiefly to the quadriceps group. The patient is unable to extend leg on thigh even to a right angle, owing to the strongly contracted hamstring muscles. There is no special atrophy below the knee. The foot is held in a slightly equino-varus position.

Knee-jerks were not obtained, nor were the triceps and biceps reflexes. No electrical examination was made.

Fritz (Fig. 2), aged nine years. Muscular wasting well marked, yet somewhat obscured by a considerable development of subcutaneous fat. His chest is flat, his abdomen protrudes. His face is brighter than that of his brother. The muscular power of the face seems intact. His pupils react normally. The pectoral wasting is marked. There is considerable wasting also in the deltoid, infraspinatus and triceps. The shoulders slide up easily. Patient can raise both arms above head; has no contracture of arms; can extend arm at elbow fully.

In the legs there is marked wasting of the quadriceps group only. He extends the leg at knee, but with no power; has as yet no contracture of hamstrings. Knee-jerks not obtained.

The patients were seen again in August, 1901, when there was little change noticeable in their condition. The younger showed slightly more atrophy but retained still considerable subcutaneous fat.

Edward, the elder, died in March, 1902, of an intercurrent infection. No autopsy was obtained.

# REMARKS ON PRIMARY NEUROTIC ATROPHY (CHARCOT-MARIE-HOFFMAN TYPE), WITH REPORT OF A CASE IN WHICH THERE WAS EXCESSIVE INDULGENCE IN TEA AND COFFEE.[1]

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The clinical varieties of progressive muscular atrophy are not as yet sharply defined. Within the last few years a number of facts have accumulated, showing the difficulty of differentiating distinctly the types to which certain forms of muscular atrophy belong. Symptoms which were considered characteristic of one form are now found to be present also in another, as for example, the reactions of degeneration, fibrillary twitching, or the mode of development of the atrophy.

The progressive muscular atrophy described by Charcot and Marie, and later by Hoffman, presents a variety which is difficult to classify, as the pathological findings are not identical in every case. The clinical picture of this form presents partly symptoms of Aran-Duchenne's disease, and partly symptoms of myopathy. However, owing to the fact that the initial symptoms are in the majority of cases identical, namely, atrophy of the peroneal group of muscles, the disease can be considered as a separate form. Moreover, if the spinal changes have not always been found to be absolutely identical, changes in the peripheral nerves have been observed in the majority of cases. The name neurotic or neuritic atrophy is justified, but it is doubtful whether the nature of the affection is always primarily in the peripheral nerves.

In cases which present spinal changes in addition to those of the peripheral nerves, it is extremely difficult to determine which of the two was the initial lesion. For this reason, the term spinal-neuritic atrophy would be more appropriate. In fact, the clinical picture of the majority of cases shows an almost simultaneous onset of the atrophy in the lower extremities and in the small muscles of the hand, thus imitating the picture of the Aran-Duchenne's type for the upper extremities. Charcot and Marie did not believe in the neuritic origin of their disease, but the cases reported later show by microscopical examination that the disease is both myelopathic and neuritic. The few cases recorded with total absence of spinal changes, or with very slight changes in the cord (Dubreuil[2]) do not, in my opinion, exclude the possibility of a spinal involvement, as it is probable that the patients did not live long enough for the spinal changes to develop.

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[1] This paper was read and the patient was exhibited before the Philadelphia Neurological Society, February 24, 1903.

The case I am about to report is an example of this spinal-neuritic form of progressive muscular atrophy. The particular interest lies in the associated symptoms and in the history. It also shows the difficulty in classification.

Mr. S. T., aged thirty-seven, gives the following history: For twenty years he had been in charge of a grocery store, where he worked daily from 5 A. M to 12 P. M. He was constantly exposed to cold. In the winter of 1897, for the first time he felt pain in the toes of both feet. The pain was accompanied by a burning sensation. The suffering at times was so intense that he had to give up work. Application of heat would make the condition worse. For four and one-half years the only symptom was pain.

Three months ago he noticed that in walking he had to raise his feet very high, and that he could not flex the foot upon the leg, especially on the right side.

*Status praesens:*—Inspection shows the legs emaciated, especially in the lower halves. The muscles of the thighs are also somewhat wasted, and the adductors are flabby. The calf muscles of both legs, and especially those of the right, are soft and flabby and give the impression of the pseudo-hypertrophic type of myopathy. The gluteal muscles are flabby. The feet are cyanosed and present the equino-varus type of club-foot. The patient stands on his metatarso-phalangeal articulations, the second and third phalanges are markedly bent downwards. The foot-drop is more marked on the right side than on the left. The nails have suffered considerably in their nutrition; some of them are absent and some are retracted.

The motor apparatus shows the following disturbances: Gait is ataxic and presents the typical high steppage, especially in the right limb. Station with closed eyes is only somewhat impaired. The knee-jerks are totally absent, even with reinforcement. The tendon of Achilles reflex is also absent on each side. No ankle-clonus, no Babinski sign obtained. Fibrillary twitching is present in the muscles of both legs, in both the wasted and the apparently non-wasted muscles. Sensations are normal to pain, but hypesthesia to touch is found, and some delay is noticed in the temperature sense on each foot, but above the ankles it is normal.

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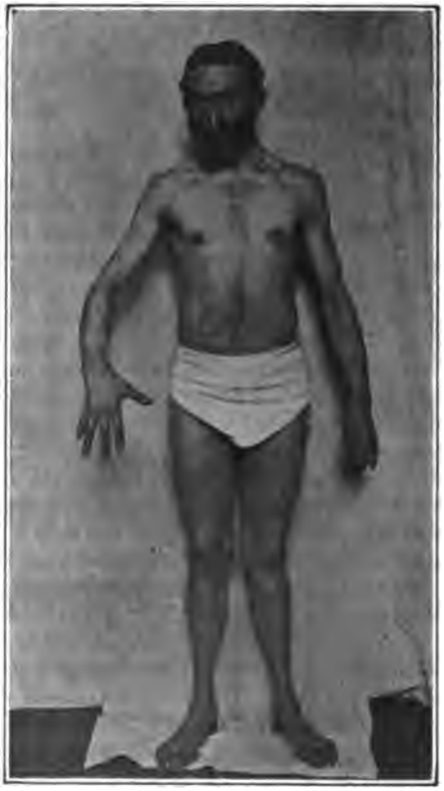
[2] Review de Méd., 1890.

The upper extremities present some flabbiness in the muscles of the arms, but apparently no atrophy. The muscles of the first interosseous space of each hand are atrophied, also in a less degree the thenar and the hypothenar muscles of each hand. There is also ataxia for fine movements in each hand; and the patient buttons and unbuttons his clothes with difficulty. There is also some diminution of power in the hands. Sensations of the upper extremities are normal. The sphincters of the bladder and rectum are normal. Sexual weakness has existed for the last two or three years.



Photograph of the patient, showing especially the emaciation of the lower limbs.

The pupils are equal, they react normally to light and in accommodation. No Argyll-Robertson pupil is found. The eyegrounds show an incipient degeneration of both optic nerves. Nystagmus is distinctly present when the eyes are turned to the right, but only slightly when turned to the left. The personal as well as the family history is negative. He denies venereal diseases and alcohol. He indulged considerably in tea and coffee; the average daily quantity used to be about one hundred ounces a day of tea, and forty ounces of strong coffee.

He complains of pain and numbness in both feet. The electrical examination gave the following result:

The peroneus longus, tibialis anticus, extensor hallucis longus muscles present distinct R. D. under galvanic current, and no response to the faradic current.

The extensor digitorum brevis, and interossei pedis dorsalis muscles show that the CaCC=AnCC on the right side, and CaCC only slightly greater than the AnCC on the left side for the galvanic current, and marked diminution for the faradic electricity. The gastrocnemius on the right side gives very little response to the faradic and galvanic currents, but no R. D.; on the left side slight diminution of electrical contractility. The adductor muscles, the sartorius and quadriceps on either side present diminution of contractility to both currents. The first interosseus space on either side presents very marked diminution of faradic contractility and CaCC is but very slightly greater than the AnCC. The thenar and hypothenar muscles also present slight diminution of electrical contractility.

To sum up, this is a case of progressive muscular atrophy, affecting mostly the peroneal group of muscles. It also shows an involvement of some small muscles of the hands, thus reminding one of Aran-Duchenne's disease; it also reminds one of the pseudo-hypertrophic myopathy by the consistence and appearance of the calf muscles. The muscles which are distinctly wasted show marked diminution of electrical contractility and partial R. D. Total loss of deep reflexes in the lower extremities, fibrillary twitching of the affected muscles, nystagmus and incipient degeneration of the optic nerves, complete the clinical picture of the case. As a coincidence, or perhaps as a cause and effect, I mention the excessive indulgence in tea and coffee.

In comparing this case with those recorded in the literature as primary neurotic atrophy, we find here also an involvement of the upper extremities in addition to that of the peroneal group of muscles. There is also the equino-varus of the feet, the fibrillary twitching, and the complete or partial R. D.

As to the deep reflexes, although in some cases they were found to be exaggerated, in others however they were diminished or lost. My case therefore can be placed alongside of the others with the same symptomatology.

There are, however, a few points which deserve attention. The history gives no trace of a similar disease in any member of the patient's family, while one of the characteristic features of the affection is the existence of the disease in the family. The age at which the disease begins is usually earlier than in my case. Finally, the presence of the nystagmus and changes of the optic nerves are rather unusual.

On the other hand, the equino-varus, the scoliosis, the total loss of deep reflexes, ataxic gait and nystagmus are symptoms characteristic of Friedreich's disease. Finally, the condition of the eye-grounds, the absence of knee-jerks and tendo-Achillis reflex, the paresthetic disturbances in the lower extremities, and the marked impairment of the sexual function, will direct our mind toward tabes.

The case is instructive from the point of view that the line of demarcation between various forms of progressive muscular atrophy cannot be maintained. The latter disease comprises a certain number of types which may differ from each other in the etiology and in the clinical expressions, but they merge into one another and have one underlying basis, namely, a degeneration of the motor neurone, or only of one portion of it, and of the muscular fibers in which this neurone ends. None of the forms possess characteristic features, which would be considered as pathognomonic.

The resemblance of the case to tabes is not unusual, as the type of muscular atrophy under consideration may be accompanied by tabetic symptoms. There are on record cases in which in addition to the degeneration of the anterior roots and anterior horns a degeneration of the posterior roots and columns also was found.

A peculiarity of the case lies also in the fact that tea and coffee were used to a large extent.

In April, 1901, I[8] exhibited before this Society a case of tea-intoxication with symptoms of ataxic paraplegia. In discussing the pathogenesis of the cord lesion, I expressed the opinion that the theine, the essential oils of the tea, and the black lead which is used to give a bloom to black tea, are at least the three main ingredients of this beverage that are poisonous to the nervous system. Similar to alcohol, lead and mercury, they are apt to produce spinal symptoms or symptoms of diseases of peripheral nerves. If not directly, they may produce a toxic condition, so as to predispose the peripheral or the central nervous system to various diseases.

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[8] Therapeutic Gazette, July, 1901.

The disease from which my patient is suffering began with a neuritis, as he experienced severe pain in the toes. The being obliged to stand in a grocery store in cold weather, a work which he had been doing for over twenty years, is not sufficient to cause a degeneration of the peripheral neurones, as examples of exposure without a neuritis are abundant. A special predisposition or vulnerability of the nerve elements is necessary. Is it not likely that the poisons of the tea and coffee which the patient absorbed in large quantities for a number of years, exercised a degenerative effect upon the nervous system and produced the spinal-neurotic atrophy he presents?

The rôle of intoxications (A. Gordon[4]) of any sort upon the nervous system is an established fact, but all the cases reported have reference to mineral poisons, while the effect of the beverages we are absorbing daily as articles of food has not yet been studied in all its phases. The importance of recognizing such a potent element as a causative factor in organic diseases of the nervous system has not yet been adequately realized. The therapeutist will perhaps be able to draw some practical conclusions from the history of this case.

I wish to express my indebtedness to Dr. John Wanamaker 3d for his assistance in taking the history of the case, and to Dr. Arnold for the photograph.

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[4] "The Rôle of Intoxications in Diseases of the Spinal Cord." Phila. Med. Jour., 1901.

# Society Proceedings.

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## NEW YORK NEUROLOGICAL SOCIETY.

February 3, 1903.

The President, Dr. Pearce Bailey, in the chair.

*Paradoxical Pseudo-Hypertrophy in Infantile Cerebral Hemiplegia.*—Dr. L. Pierce Clark presented a boy of ten years with a family history of epilepsy, insanity, alcoholism, rheumatism and tuberculosis. The personal history was good, except that when five years old he had suffered from a severe attack of smallpox. This had been followed by infantile cerebral hemiplegia, the convulsions being general and lasting for hours. A left hemiplegia was noticed the day after the convulsion. Three months after the initial stroke he had status hemiplegicus unilateralis attended by high fever. There were 47 grand mal fits in four hours. At the time of presentation there were from three to five epileptic attacks daily with an epigastric aura. Hypertrophy of the calf on the paralyzed side was first noticed three years ago, but the left arm was moderately atrophic. The ankle also showed bony hypertrophy, a still rarer condition.

*A Case of Myoclonus.*—Dr. Clark presented a Jewish boy of eleven years having a negative family and personal history. Two years ago, without known cause a spasm developed in the left biceps, triceps, pectoralis major, latissimus dorsi and sternomastoid muscles, and in a few weeks the same muscles on the right side became involved. The spasm was clonic in character and occurred from twenty to forty times a minute, with never more than a few minutes of entire freedom from the spasm. In a few months the muscles of the pelvic girdle became affected, particularly the glutei, quadriceps and sartorius. Arsenic had been of no avail in suppressing the spasms. No spasms had been observed in the fingers or toes. The musculature was good, and there was no atrophy or sensory change. All the reflexes were normal. The diagnosis of myoclonus was made on account of the bilateral, clonic, lightning-like character of the spasms of the proximal muscles.

*A Case of Multiple Neuritis with Intact Reflexes.*—Dr. Clark also presented this case, a child of five years, an imbecile and possibly epileptic. The boy had apparently had an attack of meningitis last July. He now presented the high-stepping gait and the exaggeration of all the reflexes. There was a marked diminution in the response to the faradic current. At present, he was in the recovery stage of multiple neuritis, apparently of lead origin.

Dr. J. Ramsey Hunt asked if in the case of myotonia there were a history of fright; also if the term, myoclonus, was used in the generic sense. The case had much in common with Henoch's chorea.

Dr. W. M. Leszynsky asked on what basis it was concluded that the multiple neuritis was due to lead. The distribution corresponded more to that of alcohol, and from the fact that the child had been taking beer, the probability was that it was an alcoholic neuritis.

Dr. Clark said that he looked upon the case of myoclonus as of the Friedreich type. The history of fright usually had to be taken with a good deal of allowance. He did not think it could be of the choreic type referred to, because muscular atrophies occurred and ran a rapid course. The condition was not improved by the usual treatment for chorea. The case of multiple neuritis was first thought to be one of alcoholic origin, and an effort was made to obtain such a history, but the history of beer drinking was not at all satisfactory or definite. The child had been playing around a cellar and had freely dabbled in lead paint a short time before the illness.

Dr. Pearce Bailey thought the sharp, shock-like electrical contractions of the muscles and the peculiar sound emitted by the boy should lead one to think of the type of electrical chorea.

*A Case of Myokymia.*—Dr. R. M. Daly presented a man of twenty-eight, a printer by occupation. There was no history of lead colic, and no family history bearing upon the case. For years he had engaged freely in athletic sports. About one year before coming under observation, after having walked for eleven hours, the calf muscles began to twitch. When first seen, last October, his general condition was good, as were also his habits. His gait and station were normal, and there was no paralysis of the muscles. Facial innervation was more marked on the right side. There was a fibrillary twitching of all the muscles of the legs and of the posterior muscles of the thigh. Sometimes the same bundle of muscular fibers would contract several times in succession. A blow upon the muscles brought out the contractions well. The electrical contractions were negative. There had been no evidence of atrophy during the time the patient had been under observation. There was no disturbance of the special senses, and the superficial reflexes were undisturbed. The knee-jerk was increased; there was no clonus or Babinski reflex. He had received increasing doses of strychnine and arsenic, and had received galvanic and X-ray treatment, but all without benefit. Quite recently he had been placed on gelsemium, and the twitchings had diminished somewhat.

Dr. C. L. Dana said that while the condition was not important he thought it was of interest to recognize it as a distinct symptom. Fibrillary twitchings were common enough in states of exhaustion of any kind. Myokymia occurred in neurasthenic states, and sometimes also in alcoholics.

*Non-Septic Cerebritis.*—Dr. Harlow Brooks read this paper, reporting a case. He said that these cases had not been frequently reported in recent medical literature, and hence, the case he had seen seemed to be worthy of reporting. The disease was not so very rare, but it was the exception for it to be recognized because of our scanty knowledge regarding it. Microscopical examination in a considerable number of instances had been found to be the only certain method of recognizing the condition. A considerable number of cases recovered wholly or partly. The cases going on to recovery were apt to be set down as examples of hysteria. Metabolic poisons and those toxins produced by the growth or action of bacteria should be just as competent to produce inflammation as injuries. In all inflammatory diseases the question of the occurrence of hemorrhages depended upon the severity of the inflammation and the situation of the lesions. He had no sympathy with those who insisted that an inflammation was necessarily dependent upon bacteria or their products. Acute encephalitis might terminate in death if the disease involved parts of the encephalon essential to life. When complete recovery ensued it was probable that the inflammatory products were almost completely absorbed. When there was only partial absorption there would be more or less impairment of the functions of the brain. Such a condition not infrequently occurred in alcoholics or after severe attacks of insolation. The following case was then reported: The patient was a girl of nineteen years, by occupation a domestic. There was a history of headaches and bilious attacks. She was naturally rather dull. On the night previous to her entrance to the hospital she appeared to be as well as usual, but was found unconscious the next morning, and was thought to be suffering from poisoning. On admission, she was unconscious, and the respirations were stertorous. In response to pin-pricks the hands and feet were withdrawn. The pupils were moderately dilated and reacted somewhat irregularly to light. She was unable to swallow and did not respond to the usual forms of stimulation. Kernig's sign was absent. On October 21, or two days later, the urine was normal except for a trace of albumin. The temperature varied between 100° and 101°F. Two days later some casts were found in the urine. On October 25, a bedsore appeared to be forming over the sacrum, and the temperature was rising. There was a leucocytosis of 14,000. On October 27 the patient took two ounces of water by mouth, but with this exception all nourishment had been by rectum. On October 30 she spoke once, as she had done on the previous day. The bedsore was getting rapidly worse. On November 1 she spoke rationally and complained of pains all over. Some nourishment had been taken by mouth. She was very uneasy during the night. On November 6 the temperature varied between 102° and 105° and the pulse between 140 and 160. She died with a temperature of 106°F.

Several blood cultures were taken before death, and these proved negative. An examination of the eyes was also negative. The necropsy was begun two hours after death, and was conducted by Dr. Janeway. Nowhere in the body was there any inflammation except, of course, in the bedsores. Slight cerebrospinal meningitis was found of the cellular type. There was also a general non-septic cerebritis, most marked in the cortex and particularly in the motor area. There was degeneration of many of the ganglion cells in the cortex, apparently of recent origin, and of many of the fibers arising from the large pyramidal cell layer of the cortex. There was diffused degeneration of many of the fibers passing through both internal capsules, and limited to no particular portion of the capsule. There was inflammation of the tissues of the cerebellum, though to a much less degree than in the cerebrum. There was degeneration of many of the descending fibers of the pons and medulla. There was also degeneration of the chief descending tracts of the spinal cord and of some of the fibers in the ascending tracts. There were cytoplasmic changes in the ganglion cells of the anterior horns of the spinal cord. The examination showed the case to be one of organic and not functional disorder. It seemed safe to assume that the cause was not an infection, and the almost unavoidable conclusion was that the lesions were of toxic origin, possibly bacterial. The patient had been in the habit of taking headache powders, and it was possible that this was responsible for the cerebritis.

Dr. J. Arthur Booth said he had seen this girl a few hours after her admission to the hospital, and had also been present at the autopsy. She was deeply comatose, and there was absolutely no previous history. The pupils were moderately dilated, and they reacted to light. There was absolutely no response to pin-pricks except in the feet and hands. He had been unable to make a diagnosis. At the autopsy the brain appeared to him normal except that it was rather soft.

Dr. B. Sachs preferred to speak of this condition as a non-suppurative encephalitis. He had tried in a number of instances to recognize these cases and differentiate them from other cerebral conditions, particularly from apoplexy. However, he had found it extremely difficult to make the differential diagnosis. In children there was a special type in which the patient passed slowly into a state of unconsciousness, and remained in this state for some days or weeks, when consciousness was slowly regained. After this the most striking symptom was a persistent aphasia, though the general clinical picture was that of a cerebral hemiplegia.

Dr. W. M. Leszynsky said that he had had an opportunity of examining this girl about one week before her death. At that time her condition was similar to that described by Dr. Booth. He learned that the rise of temperature had been coincident with the progress of the extensive sloughing bedsore, and it was evident that the patient was then suffering from general sepsis. He was disposed to think that this condition of general sepsis must have had considerable influence upon the post-mortem findings. He recalled a case in which a woman had remained unconscious for three months, requiring to be fed by the stomach tube during this time. Her condition was supposed to be due to ovarian trouble, and as at that time the fashion was to remove the ovaries for slight cause, both of her ovaries were removed without much benefit. She regained consciousness after the operation, and described her condition during the unconscious state. She died shortly afterward, and the post-mortem findings failed to throw light upon the nature of the case.

Dr. Joseph Fraenkel said that the existence of non-purulent encephalitis had been proved in the monograph of Oppenheim. He would adopt Oppenheim's teaching with regard to these cases. The absence of the usual etiological factors, the presence of only very slight focal symptoms, and the presence of fever should lead one to think of the existence of non-purulent encephalitis.

Dr. Joseph Collins said that he was inclined to diagnosticate acute hemorrhagic non-purulent encephalitis rather frequently—indeed, it was rare for a week to pass without this diagnosis being made. It did not seem to him that there was very great difficulty in making the diagnosis. For instance, a child of two and one-half years had been seen at his clinic that day. A short time ago it had become dull, inactive and irritable, and somewhat feverish. On the second day there was a convulsion lasting three-quarters of an hour, most marked on the left side. After the child had remained stupid for ten or twelve days it gradually regained its normal activity. On examination, there was a condition of hemiparesis with normal mentality. This case seemed to him a very clear one of non-suppurative encephalitis. In the class of cases referred to by Dr. Sachs he had never seen any rise of temperature, nor was there that degree of recovery which seemed to him so essential to justify the diagnosis of non-purulent encephalitis. He thought it was about as frequent as pneumonia, and the analogue of it. He had very grave doubt about the case reported by Dr. Brooks being one of non-suppurative encephalitis. There must have been just as great changes in the spinal cord as in the brain, otherwise there would not have been such a rapid and extensive development of bedsores. It was possible that there was present some intoxication that had not been revealed by the investigation, and which might explain the symptoms and the cause of death in some other way. The clinical description seemed to point to encephalomyelitis.

Dr. R. H. Cunningham said that he had seen two somewhat similar cases. One of them was in Mount Sinai Hospital many years ago. The history was that the young woman after a disappointment in love had begun to be sleepy and stupid, and then became unconscious. Pin-pricks or faradic stimulation of the skin caused some response at first, but in a few days she became deeply comatose. The temperature was moderate for a few weeks, and then it suddenly rose and death soon ensued. The autopsy findings were negative. He had examined some sections of the brain and spinal cord, and had found leucocytes surrounding the blood vessels. The Weigert method showed absolutely no change. The diagnosis at that time was sub-acute cerebritis. In 1895 he had seen a similar case, occurring in a school girl. The symptoms and course were about the same, but death occurred in about two weeks. The autopsy findings here were also negative. The microscopical examination of the brain gave exactly the same results. These findings were certainly not characteristic of any one disease.

Dr. Brooks said that undoubtedly the patient became septic from the large bedsore, but this was so shortly before death that the autopsy did not show the changes which were ordinarily observed in the organs under such conditions. On admission, the patient had fever, and the bedsore did not develop for a week or ten days afterward. If there were a septic condition arising from the bedsore there would have been a general infection of the blood, and various micro-organisms would have been found in the blood, yet a very competent bacteriologist failed to find any such condition. All of the classical stages of inflammation were represented in the brain. He had made careful sections of the cord, but had been unable to find the inflammatory process which one would naturally look for as the cause of the bedsore.

*Case of Myoclonus Multiplex of Friedreich.*—Dr. J. Ramsey Hunt reported a case of this kind that he had seen in July, 1901, together with the autopsy findings. The man was forty-five years of age, a tailor by occupation, and a man of good habits. A few months before admission to Bellevue Hospital one knee had been operated upon for tuberculosis. The peculiar tonic contractions of the muscles began some months previously. There was a history of despondency from financial straits. The muscles of the arms and legs, and of the shoulder girdle showed very active twitchings. The whole muscle would bound forward as if by an electrical stimulation. No tetanic contraction was detected. The face muscles showed none of these contractions. The latter were most active when the patient was recumbent. The isolated contraction of individual muscles, such as the supinator longus and the sartorius, was the most noticeable feature. Sensation was undisturbed. The tendon and skin reflexes were very active. There were no stigmata of hysteria, and no evidences of mental impairment. A few weeks later he developed fever, and died apparently of general tuberculosis. The myoclonus persisted throughout the acute febrilic attack and for fifteen minutes or more after cardiac pulsations had ceased. A careful examination was made of the Rolandic and other areas of the cerebral cortex, of the spinal cord at different levels by all of the usual methods, but with negative results. The affected muscles showed an unusually marked hypertrophy. The transverse striations were normal. In some places a central position of the nucleus of the sarcolemma sheath was noted.

*Case of Choreic Tic, with Remarks on the Classification of Myospasms.*—Dr. Charles L. Dana read this paper. He said that apparently no case had been observed in England, Scotland or Ireland, whereas 14 cases had been reported in this country, and two other cases had been reported under a different name. After carefully reading the descriptions he had come to the conclusion that no case of paramyoclonus of Friedreich had been reported in this country, not even the case just reported by Dr. Hunt, although it came nearer to that type than the others. The speaker then reported a case of paramyoclonus like those that had been published by other American observers. The patient was a man of forty-two with a history of the mother having had similar attacks during the time she was pregnant with this son. The man said that he had been healthy until the age of eleven years, when he had been rendered unconscious by the kick of a horse. Shortly after this the first attack of myoclonus developed. The trouble continued with various remissions for the 23 years which had elapsed before coming under Dr. Dana's observation. When first seen he was nervous, anemic and badly nourished. There was no disorder of the sensory sphere. When the attacks came on he was seized with rhythmic movements of the head, and the hands and feet quivered. During the attack there was considerable palpitation of the heart; he perspired profusely and became exhausted. These contractions came on at very short intervals, and were apt to last for several days unless he took some narcotic, when the attack would cease. The patient had not been improved by any treatment he had employed, including hypnotism. A short time ago the patient reported that his general health was much better, and that the attacks were milder and less frequent. Myoclonus multiplex, Dr. Dana said, occurred also in connection with epilepsy. Mention was made of a case of myoclonus that had been under observation for a number of years. The attacks had developed after an operation on the thyroid gland following an abscess. The contractions were both clonic and tonic, and involved practically the whole musculature of the body. He looked upon the case as a mixture of chronic chorea and tic. Myoclonus occurred in association with epilepsy, chorea, tic and paresis; it also occurred in family forms. All three types of myoclonus were distinctly degenerative types of mobile spasm. There was another group of myoclonus multiplex which occurred in the form of recurrent spasmodic attacks, although sometimes the attacks were continuous. The trunk muscles and the proximal segments of the body were chiefly affected, and the contractions were very violent and of a rhythmic character. These cases had been called hysterical myoclonus, but in the cases he had seen there had been no distinct evidence of hysteria. Fright or shock was a common etiological factor; his own case was of the family type. The cases reported by American observers approached more closely to a disease entity than did the case of Friedreich. He was inclined to think that the case of Friedreich belonged more properly to the class designated as myokymia. There had been reported cases of paramyotonus which ran a course quite comparable with that of paramyoclonus.

Dr. B. Onuf said that it was difficult to determine from the clinical reports found in the literature whether the muscles were hypertrophied or atrophied, and the subject was still further confused by the variations in the muscle fibers resulting from the use of various hardening fluids.

Dr. L. Pierce Clark believed that Dr. Hunt's case was a typical one of myoclonus. It was very evident that others had not met with Friedreich's type. In some recent experiments the injection of carbolic acid into animals produced a condition of myoclonus multiplex. Recovery almost never took place in a well marked case. The fact that over half of the cases reported had been associated with some form of disease, especially cortical, would seem to place the disease as cortical and not muscular. The treatment that he had found the most valuable consisted in the alternate use of bromides and chloral, with venesection and hypodermoclysis or enteroclysis at the time of the attacks. In many respects the symptomatology of myoclonus simplex and that of status epilepticus were similar.

Dr. Hunt said that the examination of many supposedly healthy muscle fibers had failed to reveal any like those in his case, the fibers in the latter being almost double the size of normal ones. It was not mentioned as an essential, but as an interesting finding. In his opinion, the distinguishing feature of myoclonus of the Friedreich type was the occurrence of contractions of individual muscles not under the control of the will except in conjunction with other muscles in the performance of co-ordinated acts.

Dr. Dana said that it was evident that we were dealing with two very widely different diseases, one represented by the case reported by Dr. Hunt, and the other, the myoclonus epilepticus described by Dr. Clark, and characterized by very violent contractions and coordinate movements. Dr. Clark had shown that the myoclonus of epilepsy was distinctly a cortical disease.

# PHILADELPHIA NEUROLOGICAL SOCIETY.

February 24, 1903.

The President, Dr. H. A. Hare, in the chair.

*The Changes Found in the Central Nervous System in a Case of Rabies.*—This paper was read by Dr. C. L. Allen. (See page 280.)

Dr. D. J. McCarthy said he had examined some of the sections from this case, and the lesions were minor as compared with those found in the dog, and were much less than those which he had seen in three other cases in the human being. In the present case, however, the inoculation experiments rendered the diagnosis positive. The presence of lesions in the cells of the capsules in the spinal ganglia in a case like this, with a history of alcoholism, would not be sufficient to render the diagnosis certain. The clinical symptoms in the case reported were not those of hydrophobia. In the three cases that he had seen there had been prodromal symptoms, followed by fever with a temperature of 103 deg. F., and symptoms of bulbar irritation. There was no special fear of water, but when the attempt to swallow was made the spasm would start in the throat and extend to the neck muscles, and afterwards to other parts of the body. In the interval there was depression. After a day or two delusions developed, and the case went on rapidly with heart failure and death. In none of the human cases was there the paralysis which is rather typical in the lower animals and which is of an ascending type.

Dr. William G. Spiller said that he had examined the specimens from Dr. Allen's case, and that the lesions were those which are found in many infectious diseases. He thought that it was quite possible that in this case the symptoms were modified by the condition of alcoholism. From the results of the experimental work he thought it probable that this was a case of rabies.

He called attention to some observations which he had made a year or two ago. At that time only one or two investigators had referred to similar lesions in any condition except rabies. These lesions have since been found in other diseases than rabies.

Dr. William Pickett referred to the mental aspects of these cases. There is no insanity of rabies. Rabies, like other infectious diseases, is commonly accompanied by delirium. This begins in the prodromal stage, grows violent in the stage of maximum intensity, and in the third, or paralytic stage, subsides.

In the past year three cases have been admitted to the detention wards at the Philadelphia Hospital as cases of rabies. Two of these were young men with hysteria major, the third an imbecile girl, who "barked like a dog," though never bitten. The men had both been bitten, and later had become addicted to alcohol as a result of morbid fear of developing hydrophobia. In both cases he attributed the hysteria major to the alcoholism superimposed upon the nervous excitement set up by the fear of the disease. This mental excitement is preferably spoken of as delirium instead of as mania. His own view was that in Dr. Allen's case the delirium of alcoholism and of hysteria was mingled with that of rabies. The symptom of barking like a dog, and the fact that the patient became lucid for a time, would support this view.

Dr. H. A. Hare asked whether the occurrence of posterior paralysis, coming on in the animal after inoculation, were of positive diagnostic value. His own experience with animals was that many poisons, when injected, are likely to produce posterior paralysis. In 1888 he had seen at the Brown Institution in London the deer sent from the Queen's preserves in Richmond Park for study. They developed posterior paralysis, but only after several days of delirium, in which they were mad, biting at their cages, acting in a manner quite opposite to their normal character. He raised the question, whether or not posterior paralysis without other symptoms possessed much value as a diagnostic aid in determining the presence of rabies.

Dr. Charles Lewis Allen said that in the case reported the clinical history was very fragmentary, and that he had not himself had an opportunity of examining the patient. With regard to the ascending paralysis, he had simply accepted the report of Dr. Ravenel, who had not entered into any details but stated that the clinical picture presented by the inoculated rabbits was typical of rabies.

*A Case of Traumatic Facial Paralysis Treated by Nerve Anastomosis. A Preliminary Report.*—Dr. Harvey Cushing, by invitation, reported this case. The patient, a young man of thirty, was brought to the Johns Hopkins Hospital, March 29, 1902, having received a bullet wound posterior to the right ear, destroying the mastoid process, middle ear, and paralyzing the facial nerve. Six weeks later an effort was made to restore function in the paralyzed muscles by anastomizing the paralyzed nerve with the spinal accessory, whose motor territory was sacrificed for this purpose, the nerve being divided *in toto* and brought into relation with the main trunk of the injured facial.

By the middle of August (95 days after the operation) the patient was found to have regained considerable control over the facial muscles. The movements at that time, however, were not coordinated, and were occasioned chiefly in association with volitional movements, which called the trapezius and sternomastoid into play. Thus, for example, shrugging the shoulder or elevating the arm as well as rotating the head to the opposite side would produce a general, though not a pronounced, hemilateral contraction of all the facial muscles.

Soon, however, coordinate action of the facial muscles began to be appreciable, so that for instance the eye could be closed fairly well, or the lips puckered in an effort to whistle without calling forth associated shoulder movements or contraction in the other muscles of the face.

In October (168th day) faradic response was obtained for the first time in the facial muscles on direct stimulation. Not, however, until the 207th day was it found possible to elicit contractions from stimulation of the nerve.

At present, nine months after the operation, the response from both direct and indirect stimulation of faradic and galvanic currents is practically equal on the two sides, the only difference being an anatomical one, namely, that the point from which stimulation of the nerve could be produced on the operative side was two or three cm. lower than on the normal side, owing to the dislocation of the nerve by the operation. The paralysis of the face at this time in repose is practically unrecognizable. On volitional effort the eyes can be closed and the mouth moved with almost complete symmetry. The associated facial movements, when the shoulder (trapezius) and head (sternomastoid) are vigorously moved, still persist.

So far as Dr. Cushing was aware, this operative procedure has only been twice described in literature. The first case was that operated by Faure in 1898, 18 months after the facial had been paralyzed by injury. The result was unsuccessful, probably owing to the long duration of the injury. The second case was operated upon by Kennedy, who intentionally divided the facial nerve for spasmodic tic and made an anastomosis with a result, which corresponds in all respects to that which has followed in Dr. Cushing's case. In consideration of the distressing and obtrusive deformity, which is resultant to paralysis of the N. facialis, it is a matter of some surprise that more attempts have not been made to restore function in these cases by following the lead which Faure's description must have given.

The most interesting feature of this transplantation of nerves lies in the physiological consideration of the nerve mechanism, which recovery brings into play. In what way do volitional impulses, traveling over a new path to the motor territory whose original central connections have been severed, reach this territory? The answer to this question must necessarily remain conjectural until experimental observations on the higher apes have helped to solve its complexities. Several theories are worthy of consideration. It is possible, on the one hand, that the cortical centers concerned in shoulder movements and in rotation of the head may in the course of time be educated by long training so as to coordinate the impulses which have been sidetracked by way of the N. accessorius into the facial territory. On the other hand, cortical centers originally presiding over movements of the face may continue to play a part in the coordinate action of these muscles, possibly by influencing the higher neurones connected with the N. accessorius through the intermediation of connecting cortical tracts. It is possible also that new centers may be developed, though this would hardly be expected to take place after adult life.

The study of Dr. Cushing's case has made it seem probable that volitional and coordinate movements have, so to speak, been superimposed upon the incoordinate movements of the face, which first appeared during recovery in association with the action of those motor territories in which the trapezius and sternomastoid were normally concerned. Dr. Cushing's impression consequently was that there has been an education of the connecting fibers of the cortex between the facial centers and those associated with shoulder movements, so that the original facial centers have in the course of time assumed the function, to a certain extent, of governing these movements, the impulses reaching the face by way of the paths from cortex to spinal accessory centers in the upper part of the cord, and thence to the face by way of the transplanted nerves.

*Anastomosis of the Seventh and the Spinal Accessory Nerves for Traumatic Facial Palsy.*—Dr. W. W. Keen reported this case. E. C., age twenty-one, Camden, N. J., was admitted to the Jefferson Medical College Hospital November 17, 1902, with facial palsy. Four weeks before admission he was struck with a glass bottle, which broke as it struck him, and inflicted a deep incised wound, 4 cm. long, in front of the left ear. Immediate paralysis of the left side of the face occurred. The wound suppurated, but finally healed just before his admission.

Operation November 26.—Dr. Keen made an incision in the old scar and with great difficulty, owing to the fibrous tissue, was finally able to identify the seventh nerve in the upper part of the incision about at the level of the external auditory meatus. This was undoubtedly only the upper part of the nerve. He could not find the main trunk. He then extended the incision down to and along the anterior border of the sterno-cleido-mastoid, lifted the muscle and discovered the spinal accessory almost at its posterior border. At this point it bifurcated, one branch entering the sterno-cleido, the other passing to the trapezius. On measuring the distance from the nearest point where the nerve could be turned upward to anastomose with the facial, he found that the main trunk would be too short and would not reach the seventh. Accordingly he made a second incision at the posterior border of the sterno-cleido, laid bare the branch to the trapezius, divided it and brought it under the sterno-cleido forward and attempted to anastomose it with the seventh nerve. It was still too short. He was, therefore, obliged to split the spinal accessory toward its root for about 3 cm. This gave him a long enough piece of the spinal accessory to anastomose with the facial, which was accomplished without any tension. In order to identify the facial, he used the faradic battery, using the indifferent electrode on the shoulder and a copper wire electrode to find the nerve. At no time was he able to produce any contraction of the facial muscles by means of a current strong enough to make contractions of the shoulder. This added materially to the difficulties of the operation, for the nerve could be recognized only by its appearance and anatomical relations.

The patient made a perfectly smooth recovery and was discharged December 3, with instructions to report to the Neurological Department for electrical treatment of the muscles.

He has never presented himself for treatment. His present condition on February 21, 1903, is unchanged from what it was before the operation.

Two days after the operation Dr. Alfred Gordon made the following report as to the condition of the muscles.

*Galvanic Current.*—M. frontalis—No. RD, but the response is weak. M. orbicul. palpebr.—The CaCC is but slightly stronger than AnCC. M. levator labii super. alaeque nasi—Same. M. levator labii super and MM. zygomatici—No response to anode or cathode. M. levator menti—The CaCC is but slightly stronger than AnCC. M. buccinator—Normal, but feeble reaction. M. sterno-mastoid—Feeble reaction, but no RD. M. trapezius—Same.

*Faradic Current.*—M. frontalis—Very feeble response. M. orbicul. palpebr.—Response hardly perceptible. M. lev. labii sup. alaeque nasi—No response. M. lev. labii sup.—No response. MM. zygomatici—Response hardly perceptible. M. levator menti—Reaction very feeble. M. trapezius—Reaction very feeble.

*Conclusions.*—The MM. trapezius and sterno-mastoid are markedly impaired in their nutrition, but as yet do not present the typical RD. The M. buccinator also does not present the typical RD, but its faradic contractability is totally lost; the latter circumstance makes the prognosis unfavorable for this muscle. The MM. levators labii and zygomatici do not respond at all to either current. The M. orbicularis palpebr. presents the beginning of RD. The M. frontalis is in better condition than the others.

Dr. William G. Spiller said that astonishing as was the return of function after anastomosis of nerves, it was not quite so surprising if it were remembered that when a motor nerve is divided and immediately reunited, there is not a reunion of the same fibers, but that different fibers united so that other nerve cells were brought into association with the nerve fibers.

The question, asked by Dr. Cushing, whether or not there will be degeneration of the central system of nerve fibers after lesion of the peripheral system, depends very much upon the age of the animal. In a young animal if the peripheral system of nerve fibers is destroyed the central portion of the system atrophies or does not develop as it otherwise would. If, however, the animal is well developed and an adult the change does not take place to the same extent.

The question as to whether the operation done by Dr. Cushing is advisable or not is much easier to decide where the facial nerve has been cut, than it is in cases where there is facial paralysis, as from cold. In severe cases of this type, even where the reactions of degeneration are marked, there is often some return of power. In such a case, Dr. Spiller thought that it would be wise to wait at least three or four months before deciding to operate, but it must be remembered that in these cases of nerve anastomosis the prognosis depends very much upon whether or not the injury is recent.

*Nerve Anastomosis in a Case of Anterior Poliomyelitis.*—In this connection Dr. Spiller and Dr. J. K. Young exhibited a patient with infantile spinal paralysis on whom Dr. Young had performed nerve anastomosis. The patient was a child in whom the anterior tibial muscle alone was paralyzed, this paralysis being the result of anterior poliomyelitis.

The operation having been decided upon, the most important feature was the route to be selected. From a careful dissection personally made, Dr. Young decided that the lateral route was the better one, and accordingly an incision 10 cm. in length was made downward from the head of the tibia in the long axis of the leg. The incision included the skin and superficial fascia. The deep fascia was divided upon a groove director, exposing the peroneal nerve. The nerve was followed down, and by separating the peroneus longus muscle three divisions were found, (1) the fasciculus of the nerves supplying the upper part of the anterior tibial muscle, (2) the anterior tibial nerve, (3) the musculo-cutaneous nerve, these divisions corresponding to those found in the cadaver. It was decided to take the upper division, of which there were four or five fasciculi, and perforating the external division, the musculo-cutaneous, the former were united to the outer side of the latter by fine catgut sutures.

Great care was taken not to injure the musculo-cutaneous nerve any more than necessary, and the fasciculi of the nerves which supply the anterior tibial muscle at its upper part were pushed through the incision in the external musculo-cutaneous nerve without any attempt being made to separate the nerve fibers from the sheath. Small instruments were used, and the nerves were handled as little as possible.

The nerves which were anastomosed were divided as high up as possible, so that there would be no tension upon them. The deep facia was not closed with sutures. The skin was united by interrupted sutures. Over the antiseptic dressing a plaster of Paris cast was applied to insure fixation of the limb.

At the time of presentation improvement in the action of the anterior tibial muscle was observed.

*Myokymia of One Side of the Face.*—Dr. Spiller reported a case of this kind in which the movements were confined to the left side of the face. Bernhardt had reported a case of constant fibrillary tremor affecting the left side of the face. A similar case had come under Dr. Spiller's observation. These were the only two cases that Dr. Spiller knew of.

Dr. A. A. Eshner remarked that while it was not so difficult to understand how in Dr. Cushing's case facial motor impulses, originating in the cortical motor centers, could be carried through the spinal accessory nerve, it was most difficult to comprehend why spinal accessory impulses should not be transmitted to the peripheral distribution of the facial nerve. It seemed to him that observations on nerve anastomosis, like that reported, threw some light upon the case recorded by Drs. Harte and Stewart of complete severance of the spinal cord by a bullet, in which immediate union by suture was followed after a time by a notable restoration of motor and sensory function.

*A Case of Primary Neurotic Atrophy.*—This case was reported by Dr. Alfred Gordon. (See p. 354.)

Dr. F. X. Dercum said that the remarkable point about this interesting case was the condition of the eye-ground and the nystagmus. It was an interesting question whether or not the toxic history played a rôle in the eye-ground changes.

Dr. Charles K. Mills said that the case looked like some that he had seen where there was association of progressive muscular atrophy with tabes, although the tabetic symptoms were not in this case thoroughly developed.

Dr. Charles W. Burr thought that it was not wise to attribute the condition in this case to the excessive use of tea or coffee. This excess is very common while the disease in question is very rare. It was his opinion that it had never been proven that the use of tea produced any organic change in the nervous system. It seemed to him that Dr. Gordon's patient had disease not only of the peripheral nerves but also, at least, of the anterior horns of the spinal cord, and possibly also of the posterior part of the spinal cord.

Dr. Spiller remarked that the symptoms in Dr. Gordon's case had some resemblance to those of interstitial, hypertrophic and progressive neuritis of childhood of Dejerine and Sottas, and yet the case was lacking in some of the essential features of this disease.

Dr. Alfred Gordon called attention to the fact that in reporting the case he had simply stated that it was one in which there was excessive indulgence in tea and coffee, but he had not said that this was the cause of the symptoms. He had reported in 1891 a case of tea intoxication with spinal symptoms. It might be that as result of this intoxication the nervous system was predisposed to various affections.

With regard to diagnosis he had called attention to the fact that the case was not of the classical type, that it was a mixed case. It shows how difficult it is at times to classify the progressive muscular atrophies.

# Periscope.

## AMERICAN JOURNAL OF INSANITY

(Vol. 59, 1903, No. 3.)

1. The Insane in Brazil. **Kidder.**
2. A Contribution to the Chemistry of Nerve Degeneration in General Paralysis and Other Mental Disorders. **Coriat.**
3. Dermatoses of the Insane. **Winfield.**
4. Report of a Case of Dementia Præcox with Autopsy. **Dunton.**
5. A Case of Abscess Diagnosed as Brain Tumor. **Gordinier.**
6. A Case of Brain Tumor in a Woman Seventy-eight Years of Age. **Madison.**
7. Acute Paresis with Report of a Case; the Clinical History and Pathological Findings. **Paton** and **Rusk.**
8. On the Motor Cortex. **Farrer.**
9. A Case of Metastatic Adrenal Tumors in the Left Mid-frontal and Ascending Frontal Convolutions. **Channing** and **Knowlton.**

1. *The Insane in Brazil.*—The government hospital at Rio de Janeiro is a modern, well-equipped institution with the latest appliances for the examination of patients, which is very complete, and the latest and best electrotherapeutic and hydrotherapeutic apparatus. The chronic insane are cared for in agricultural colonies. The hospital is comfortably furnished, except the dining rooms, where the furnishings are very scanty and poor. A training school for nurses is maintained. Mechanical restraint is not used, and the patients present no marks of violence. In some of the provinces the insane are cared for in religious charitable institutions which are of a low grade. The State of Sao Paulo has a good hospital with agricultural colonies attached. There are several private institutions at the Federal capital. Considerable attention is given to hydrotherapy. The death rate of the insane is large, being due in a measure to the tardiness in sending the patients to the hospital, many dying within the first twenty-four hours after admission.

2. *A Contribution to the Chemistry of Nerve Degeneration in General Paralysis and Other Mental Disorders.*—The chemistry of nerve degeneration is at present limited to katabolic processes in the lecithin, the main constituent of the myelin sheath, and to decomposition of the axis cylinder, of which little is known. The decomposition products of lecithin are found chiefly in the cerebrospinal fluid, which latter varies under different conditions, like age and certain diseases, such as general paresis, and its color is changed under certain pathological conditions and by certain remedies; while normally alkaline, it may become acid in reaction under long-continued activity of the nervous system and is due to the lactic acid of degeneration. The toxicity of the cerebrospinal fluid is increased in general paresis, owing to the cholin and other products of nerve katabolism and reaches its maximum after the epileptoid seizures and is believed to depend on cholin, which is the decomposition product of lecithin, and is the result, not the cause of the convulsive seizures. The knowledge of phosphoric acid metabolism is slight. In degenerative conditions, like general paresis, phosphorus has been found diminished in the nerve tissue and is a measure of the degeneration. A series of 34 cases suffering from different psychoses, in which the cerebrospinal fluid was examined and the technic fully given. These cases are further tabulated as to the chem-

ical analyses, but the results do not bear abstracting and would have to be quoted verbatim to be intelligible.

3. *Dermatoses of the Insane.*— No special relation of the various affections found are proven to have any connection with the mental disorder. Of the stigmata of degeneration hypertrichosis, on which great stress is often laid, was most prevalent in females, and a case is cited of melancholia, in which with the progress of the disease the hypertrichosis became marked, and as she improved mentally the hairs dropped off, leaving the skin as free as before the illness. Telangiectasis was found to be as prevalent in the sane as in the insane. The skin was found to be no more unctuous or odoriferous than in the sane. On the whole the insane are more subject to degenerative dermatoses than the sane, yet the cutaneous stigmata are found to be of little clinical value. A tabulated statement of the findings concludes the paper.

4. *Report of a Case of Dementia Praecox with Autopsy.*—This patient was under observation four years and died of tuberculosis with the katatonic form of dementia præcox. It is of a young man of 22, clerk. Mother died of tuberculosis. The onset was sudden, patient became confused, refused food with the other symptoms peculiar to this disease type. A summary of the microscopical findings in the brain are as follows: slight cell change distributed over the entire brain, but most pronounced in the first frontal convolution. Cells present central chromolysis, an occasional slight pale yellow degeneration, slight cell atrophy, atrophy, discoloration and swelling of the nucleus; folding of the nuclear membrane and an endonucleolus. Deeper layers most affected, motor cells show slight changes like the above. Slight increase of neuroglia nuclei. Phagocytosis well marked and considerable cell degeneration. No change in the medullated fibers and no marked vascular changes. No cell shrinkage, so frequent in tuberculosis. An exhaustive autopsy report is given and the paper illustrated by four plates showing the cell changes.

5. *A Case of Abscess Diagnosed as Brain Tumor.*—The case of a man of 52, married, farmer, with no hereditary history and perfectly well until two years ago before the final illness, when he had several attacks of indigestion. His final illness began with unilateral convulsion followed by unconsciousness, in which head and eyes first turned to the left, then whole body convulsed and loss of consciousness. The effects of the attack were evanescent. Another attack occurred in 26 days and similar in character, except not followed by unconsciousness; numbness preceded neither. This one was soon followed by another; the fourth occurred two weeks later without loss of consciousness. Movements of arm weakened and awkward after the last attack with gradual failure of memory, particularly for names and dates. No severe headache, but constant dull frontal pain. No facial symptoms except slight paralysis of central type, shown by obliteration of the left naso-labial fold and drooping of the angle of the mouth; left arm partly paralyzed and held semi-flexed. No reflex accessory derangements. Cerebration retarded, no aphasia. Patient operated and nothing indicative found and the wound closed. Patient died about three weeks later, after being delirious, followed by unconsciousness. An examination of the brain revealed a tumor-like mass involving the middle third of the right central convolution and extending forward and upward, implicating the bases of the superior and middle frontal gyri, measuring 4 cm. antero-posteriorly and 3½ cm. vertically, which was found to be an abscess, whose cavity extended forward into the frontal lobe, involving the centrum ovale beneath the bases of the superior and middle convolutions. The pus was thick and contained broken down brain tissue with micro-organisms of the colon group. The following conclusions may be drawn from this case: (1) The necessity of an exploratory puncture or incision in every apparently inoperable case when symptoms are strictly localized; (2) that typical Jacksonian epilepsy may be excited by a lesion in the white matter beneath the

cortex as well as upon it; (3) the ataxia of the arm may be explained either by involvement of the sensory fibers in the white matter of the inferior parietal lobule or by destruction of a part of the fibers of the frontocerebellar tract; (4) no disturbances of sensation and the Jacksonian attacks not preceded by numbness; (5) the paresis of the left arm following local convulsive seizures due to compression and destruction of the motor fibers of the arm area; (6) that no symptoms of compression existed; (7) this case shows the value of local convulsive movements in diagnosing local brain disease, also confirms the position of the centers for conjugate movements of the head and eyes, as for those of the arm. Also proves that local convulsive seizures may be excited by a lesion in the white matter beneath the cortex as upon it. Further that the motor cells are located ventral to the fissure of Rolando. Two photographs illustrate the article.

6. *A Case of Brain Tumor.*—This occurred in a woman seventy-eight years of age. The case of a brain tumor in an old lady of 78, which was unexpected during life, presents nothing of special importance as to diagnosis, owing to deficient clinical history in this respect. A careful autopsy was made and the brain thoroughly examined. Two photographs accompany the article.

7. *Acute Paresis with Report of a Case; the Clinical History and Pathological Findings.*—A number of atypical conditions included under general paresis differing in symptomatology, as well as in the pathological changes. Only the cases which present the following features should be classed as acute paresis: (1) There should not be any prodromal period when the symptoms were of subacute or chronic type; (2) when death occurs in acute delirious excitement, the pathologist has to determine if there is a definite organic lesion sufficient to account for death; (3) when the pathological findings reveal a subacute or chronic process in the central nervous system, acute paresis is excluded. The case of a man is reported, whose disease ran its course in a few months. The family history was negative. No severe illness. Contracted lues several years ago. He first lost interest in his work, became nervous, worried, had digestive derangements. Before admission presented symptoms indicative of mental deterioration. Physical signs on admission practically negative, except marked speech defect. Memory very poor and unable to give any intelligent account of himself. Had hallucinations of hearing of a persecutory content. Gradually become more excited and noisy at times, his conversation disconnected and incoherent, no well marked grandiose ideas. About four months after admission had a series of convulsions. Had been extremely restless before, requiring restraint. After seizures the mental weakness progressed rapidly, and finally died in a little over four months after admission, only a few days after the convulsions. The authors' résumé of the case is as follows: "It is probable that cases of acute paresis are of greater rarity than has commonly been supposed. In order to form an accurate judgment as to whether a given case may be grouped under the *forme foudroyante,* it is not only essential that the subacute or chronic initial phase be excluded by the history of the case, but the acuteness or chronicity of the pathological process in the central nervous system must be carefully considered. The pathological findings reveal nothing that is essentially characteristic of the disease. We have noted in particular a nerve-cell alteration, which is in no sense pathognomonic. There is a general and not localized disappearance of fibers from all cortical areas. The neuroglia shows an increase of the cellular elements, particularly of the larger size spider cells, with but little, if any, increase of the fibers. The blood vessels, particularly the larger arteries of the cortex, are well filled with blood; in some cases this overfilling extends to the smallest vessels. Many round nuclei are found in the perivascular spaces. In most of the cortical areas, numbers of plasma cells are present. We have failed to

find them in the tissue surrounding the vessels. The character of the changes in the various organs, as well as in the central nervous system, suggests a general intoxication. The vascular changes in the cortex are not sufficient to account for the degeneration of the nerve elements." This paper is illustrated by two plates showing the condition of the various nerve cells.

8. *On the Motor Cortex.*—This paper will not bear abstracting, as it is largely a detail of study and investigation of the motor cortex. It at first gives a history of the development of cerebral localization. The substance of the paper is best given in the author's own résumé: "The motor cortex occupies the middle region of the hemispheres, intermediate between the general and special sensory areas on the one side, and the specific association or psychic center on the other. This zone is the earliest to functionate, and the central sulcus forms to give increased room for its developing elements. It has been defined by the brilliant experimental work begun by Hitzig and Ferrier, by the clinical and pathological findings of Broca, Hughlings Jackson and a legion of subsequent observers, by the intricate myelogenetic method of Flechsig, and by the histological studies of Betz, Hammarberg and Cajal. Beginning with the observation of Hitzig that the motor area embraced nearly a half of the brain surface, its confines have gradually become narrowed. It has long been assumed roughly to include both central convolutions and the paracentral. Beevor and Horsley cast a suspicion upon the motor function of the postcentral gyrus. Flechsig discovered embryologic differences which he believed justified him in considering the postcentral the seat of general incoming sensory impulses, and the adjoining precentral the seat of outgoing motor impulses. Finally, Cajal described a specific fiber distribution in the precentral convolution which, he believes, serves to differentiate it as the motor type of cortex. That a motor type of cortex exists is abundantly proven. It is characterized by its unusual width dependent upon the marked development of the medium-sized pyramidal cell layer containing the specific plexus of Cajal, by the absence of a clearly defined granular zone, and by the presence of giant cells in the sixth layer with thicker and denser radial bundles and a richer development of the overlying tangential fibers, which are of maximum density in those portions of the convolution which contain the greatest number of giant cells, both being more conspicuous in the fissure in the lower half of the Rolandic zone, and on the summit of the convolution in the upper part of the motor region. Passing beyond the limits of the motor area, superiorly, inferiorly, anteriorly or posteriorly, one observes a gradual change in the cortical type, the third layer becoming narrower, the fifth wider, and the specific elements of the sixth disappearing. This change is typically illustrated in a cross section through anterior and posterior central convolutions. The paracentral presents both types of cortex, the transition taking place below the extremity of the fissure of Rolando, although no surface-marking determines the boundary."

9. *A Case of Metastatic Adrenal Tumors in the Left Mid-frontal and Ascending Frontal Convolutions.*—A man of forty-eight had a tumor removed, involving the left kidney, which on examination proved to be a sarcoma of adrenal origin. He improved rapidly after the operation and was about to leave the hospital, when he had several severe epileptiform seizures, and in a few days another series of similar convulsions, which were immediately followed by weakened attention. Slight symptoms of paresis soon appeared on the right side. He soon displayed some hesitancy in speaking and often used the wrong word. His mental processes gradually became impaired in progressive manner, and the functions of the right extremities also became more impaired before death; he became helpless and his mental functions extremely dull, filthy in his habits and unable to appreciate his surroundings or condition. Was finally unable to swallow

fluids readily, could not express himself intelligently, manifesting an almost complete suspension of the mental functions. Finally he died in a semicomatose state with slight elevation of temperature, accelerated pulse and respiration. The autopsy revealed a tumor of 2.5 cm. in its greatest diameter in the left temporal region. Its exact location was found to be in the posterior inferior part of the swollen midfrontal lobe, just anterior to the precentral fissure and 4.5 cm. above the beginning of the fissure of Sylvius. A second smaller tumor was found in the ascending frontal lobe, between the precentral fissure and the fissure of Rolando, measuring 8 mm. in diameter. - Two similar nodules were found in the left lung, being composed of large cells imbedded in the meshes of a rather delicate connective tissue stroma, i. e., like that of carcinoma. The cells of large size and of epitheleoid character and protoplasm of delicate reticulated appearance. Two plates show the appearance of the superior surface of the brain and a vertical section through the site of the tumors.

McCorn.

## LE NEVRAXE

(Vol. 4, 1902, No. 2.)

6. Case of Lesion of Cauda Equina. Contribution to Study of Centers of Micturition, Defecation, Erection and Ejaculation (concluded). A. Van Gehuchten.
7. Experimental Study on the Innervation of the Larynx of the Rabbit. F. de Beule.
8. Gemmules in the Nerve Cells. S. Soukhanoff.

6. *Cauda Equina Centers.*—Van Gehuchten completes his study begun in the last number of this journal. He presents a very full analysis of the work done in the centers of micturition, defecation, ejaculation, etc., and gives in great detail his results in a patient with a severe transverse lesion of the cauda. The paper is extensive, and his conclusions can be here given in fragments only. He believes with Müller that the primary centers of micturition, defecation, erection, and in part also of ejaculation are largely localized in the sympathetic ganglion of the hypogastric plexus. To these centers, however, should be superimposed those located in the conus. These, in their turn, are influenced by the superior centers of the cerebral cortex.

The sympathetic centers can functionate in a manner completely independent of the cerebrospinal centers. This probably takes place, for instance, in infants in the first months of life. Then the bladder and rectum empty themselves more or less regularly automatically at more or less regular intervals, and erection may take place. This also takes place in adults in lesions of the conus or of the inferior roots of the conus.

The medullary centers, however, exercise an influence over the sympathetic centers. Thus, in infancy, cold applied to the feet may induce a reflex micturition and similar skin reflexes in adults may stimulate vesical activity. The cortical influences are of the voluntary order and are acquired largely by the educative processes. In cord lesions their cutting off temporarily disarranges the bladder, rectal and other functions, but they may recuperate and come back to their primary functional activity.

7. *Innervation of the Muscles of the Larynx.*—F. de Beule gives a lengthy historical and experimental study on this subject, treating the subject under three main heads: (a) The distribution of the laryngeal nerves; (b) the part played respectively by the pneumogastric and the spinal accessory in the muscle innervation, and the character of their fibers; (c) the determination of the medullary center of the laryngeal nerves. So far as the muscular distribution of the laryngeal nerves is concerned de Beule favors Linget's views, holding that (1) Exner's views of a double nerve supply and from the nerves of both sides is not true; (2) Longet's view

is the correct one with the addition of the part played by the middle laryngeal nerve; (3) the crico-thyroid muscle is innervated by both the superior and middle laryngeal nerves; (4) all the other muscles are supplied by the recurrent laryngeal; this distribution is unilateral, save perhaps for the arytenoid muscle. Chapter II. of his research takes up the question of the motor fibers of the vagus and spinal accessory and the relative part played by each in the innervation. His conclusions reached after an elaborate argument, historical, physiological and pathological, are (1) that interrachidian tearing of the spinal accessory nerve is followed by Wallerian degeneration in the corresponding inferior laryngeal nerve only, and a limited number of its fibers at that, from which he concludes that the superior laryngeal and some fibers of the recurrent are derived from the pneumogastric; (2) isolated rupture of the bulbar roots of the pneumogastric is followed by immediate cessation of the respiratory movements of the corresponding vocal cord, and stimulation of the common trunk of the vagus, in these animals that had survived for a period of three weeks the intracranial rupture of the spinal accessory, gives rise to movements of the glottis. On the question of the bulbar centers de Beule claims that the dorsal vago-spinal accessory nucleus is the nucleus for the innervation of the larynx. The ganglionic centers for the motor fibers of the superior laryngeal nerves are in the lower two-thirds of the dorsal nucleus of the pneumogastric, in which region portions of the cells of origin of the recurrent laryngeal are also found. The spinal accessory is located in the upper three-fourths of the same nucleus.

8. *Gemmules.*—Soukhanoff continues his studies on gemmules, in one instance using the spinal cord of the guinea pig. He finds gemmules on some of the cell bodies of the spinal cord. In an old animal the gemmules were so numerous that they completely covered the cell outline between the dendrites. In newborn children he has found them, and in the posterior horns of an adult of fifty-two years. He holds that they are not artefacts, but contributes nothing new concerning their possible function.

JELLIFFE.

## MISCELLANY.

A CASE OF TUMOR OF THE CAUDA EQUINA. Franz Volhard (Deutsche med. Wochenschrift, xxviii. 33, 1902).

The author reports a rare case, and one which is of importance for topical diagnosis. The patient, a male, and a teacher by occupation, observed early in the year 1898 that he was obliged to urinate more often and with more difficulty than usual, and that much more voluntary effort was required during defecation. In the same year a peculiar feeling of pressure manifested itself at the lower extremity of the spine, which persisted. In 1899 impotence occurred; in the summer the left foot began to waste as well as the leg, and the fatigue in the extremity caused the patient to limp. There never was any pain in the limb. The dull feeling of pressure above mentioned still persisted and became aggravated upon bending the body, coughing, climbing stairs, and upon pressure, although there was never a definite appreciation of pain. In 1900 the symptoms gradually increased. The left leg felt heavier, and the movements of the toes ceased. After treatment in one of the German watering places during 1901 the patient felt considerably better. He could walk much better, the bladder could be emptied with much more ease, and the impotence markedly decreased. This improved condition continued for almost three months until the end of the year 1901, when the symptoms again became aggravated. In addition to the previous symptoms the patient became greatly annoyed by an excessive thirst, dryness in the mouth and nose, loss of appetite and nausea. The feces became so dry that the patient often had to extract them with the fingers. Examination of the patient showed the following: Above the symphysis the bladder could be palpated as a large tumor extending to the umbilicus. Examination of the internal organs of the thoracic and abdominal cavities showed nothing abnormal. The left leg, particularly the lower portion, was from 1 to 2 cm. thinner than the right, the left ankle joint relaxed, and the muscles of the calf limper than those of the right. There was no swaying with the feet in juxtaposition. The knee-jerk on both sides was very prompt, and the cremasteric reflex very pronounced. The Achilles reflex was only just perceptible on the right side, but could not be obtained upon the left. The plantar reflex was very much diminished on both sides. Examination of the hip-joints showed that on the left side external rotation was easily performed, yet markedly less than on the right side, while internal rotation could be forcefuly performed on both sides. Extension of the leg from the knee-joint could be easily performed on both sides, but flexion was weaker on the left side. Dorsal flexion of the feet was less pronounced on the left side. No zone of anesthesia could be detected. Only the most careful tests elicited here and there a diminished or absent appreciation of tactile impressions and in the following areas: (1) At the area corresponding to the sacrum, although the latter was not painful on pressure. (2) Directly above the knee on the left side. (3) A circular area at the external aspect of left leg. In these areas, both tactile and temperature appreciation were absent. There was no real pain, but a feeling over the sacrum described as "miserable, and pressing as if a wedge were being driven into the tissues." Electrical excitability was diminished quantitatively in the paretic muscles, but no reaction of degeneration could be obtained. The author calls particular attention to the fact that in this case the diagnosis was confined to a lesion of the conus or the cauda equina, based upon accepted topical diagnosis. The location of the lesion was of particular importance for therapeutic purposes. An appended table offers the explanation that the lesion could be inferred as existing either in conus or in cauda equina. Yet the absence of sensory irritability is in contradistinction to the symptoms in lesion of the cauda equina. But the diagnosis of a tumor of the cauda equina was made, because it explained the symptom-complex rather than individual symptoms, and because a tumor of the conus would have caused extensive symmetrical paralysis in such a time. It was intended to open the spinal column, but the general condition of the patient continued to grow worse until he died during a uremic attack. The autopsy showed a glioma directly under the conus, which not only crowded the posterior roots, but which compressed the anterior ones against the spinal column, therefore, also, explaining the slight sensory disturbances.

Dinkelspiel (Philadelphia).

# Book Reviews.

LA LOGIQUE MORBIDE: L'ANALYSE MENTALE. By N. VASCHIDE and C. VURPAS, preface by M. TH. RIBOT. Société d'Editions Scientifiques et Litteraires, F. R. de Rudeval et Cie., Paris, France.

This small volume of 260 pages is a valuable contribution to the subject of morbid introspection. Many there are who, interested in psychological problems, are wearied to death by the self-analysis of their patients. This work is one of the few of its kind that has come to our attention and seeks to study this type of mental activity as an entity and to analyze its various features.

The authors propose a classification which has many interesting features. They first separate the types of mental activity into those with diminished mental analysis and those of exaggerated mental action. They class under A, general paralysis and dementia as due to enfeeblement of mental images; idiocy and imbecility as due to imperfection of the association of images and of ideas. Mania presents a diminished type of mental analysis by reason of a too rapid succession of ideas in the field of consciousness; melancholia by reason of a profound perturbation of representation of mental images and ideas; and mental confusion as due to a disarrangement of association of images and of ideas; systematized delirium in which paranoid forms of mental disease may be classed is characterized by a fixity and uniformity of the direction and rendition of mental analysis.

The book is more suggestive than convincing, but is well worth the reading. JELLIFFE.

THE MIND OF MAN. A TEXT-BOOK OF PSYCHOLOGY. By GUSTAV SPILLER. The MacMillan Company, New York.

The science of mind, the author says, must revolutionize the whole of philosophy. He holds that the determination of the nature of the mental processes and the nature of man will set at rest once and for all those discussions that have raged around a unitary conception of the universe. Physical science and mental science will then no more form two independent and hostile camps, and speculative metaphysics will cease to exist, handing over many of its interesting problems to science.

The author here gives us a volume of some 500 pages, divided into three parts, in which are considered first, the *methods* of the study of psychology, the *general analysis* of the individual features, and a *special synthesis* of the various forms of classification as applied to psychological phenomena.

In an attempt first to place this book before our readers that they may understand its general scope and limitations, we may say that the author has followed the workers in the lines of general philosophy and metaphysics more than he has those of the workers in the realm of so-called physiological psychology. This has its advantages as well as its disadvantages. Thus, on the question of introspection, after giving a complete discussion of the different opinions of various writers, he says that although, almost without exception, the testimony directly or by implicacation against introspection as a good psychological method is crushing, his reply is as follows: "Retrospection is of necessity introspection, and if, therefore, introspection be impossible our minds are absolute blanks."

The introductory chapter on method is very interesting. After roaming the general field over, discussing many authors in their method of approach to the general problem, he passes in review the various psychological schools, the reflective, the physiological, etc.

He holds that in each instance it has been found that the positive acquirements have been scanty to an undue degree, and this he attributes to the fact that psychologists have not, as a rule, put themselves in close touch with the scientific method.

In part II., which makes up the greater part of the book, the subject of general analyses is taken up, and much stress is laid upon *attention* in its relationship to sensation. The author says that sensation, images and feelings do not exist apart from attention. He then takes up the differences of attention in the normal waking state, deliberate attention, abnormal attention, and the expanding and contracting of the field of attention.

He identifies attention with cerebral change. Willing is divided from attention, and attention from willing, in that attention is always change while volition is not change, but only points to it. The relation between these two is that between being and becoming. He thus takes an entirely new point of view on this question.

In chapter III. the question of habit, memorizing of facts, and the development of automatism is taken up. Judgment, early education, and the origin of thought is very clearly summarized.

Chapter IV. deals with systems as need-satisfying, and here the general methods of thought, different facts of the combination of thought are taken up. Expressed in the author's own words he says: "Owing to the nature of the human structure, as determined by natural selection, needs tend to arise and to satisfy themselves, the methods of satisfaction being outlined in the normal and adapted organization. Under ideal conditions, given the need, satisfaction should follow at once, in which case there would be but one step in every process. Since, however, the human structure is imperfectly adjusted to its environment, the process of satisfaction or readjustment is frequently long, complicated, and unsuccessful, and that these more or less prolonged attempts at satisfaction or readjustment, embracing observation, memory, action and thought in every form, we call the process of need-satisfying."

In part III. *special synthesis* is taken up, and he believes the business of psychology to be an inquiry into the following facts: The nature and growth of needs; the range, the effectiveness, the liability to deterioration, to improvement or breakdown, which is to satisfy the needs: the process of satisfying needs; and the distinguishing forms of such processes. This is the generalization with which the author concludes his final chapter.

There is a distinct freshness and vigor in the author's presentation which jostles one as it were from the ordinary ruts of introspective psychology, although the general tendency is in this line. We can recommend it most heartily to our readers as something ingenious, fresh and suggestive. BROWN.

THE EVOLUTION OF MAN AND HIS MIND. A History and Discussion of Evolution and Relation of the Mind and Body of Man and Animals. By S. V. CLEVENGER, M.D. The Evolution Publishing Company, Chicago.

The author is known for his many works dealing with comparative physiology and psychology, spinal concussion and medical jurisprudence of insanity; and as one of the founders of the JOURNAL OF NERVOUS AND MENTAL DISEASE it gives us pleasure to welcome the present extremely interesting work. There are twenty chapters, in some 600 pages, which run from the consideration of the earliest man to the study of the highest types of social organization.

In the early chapters the author discusses prehistoric man, those of the stone age, the dwarfs, the Turanians, the Africans, the Malays, the

Aryans and the Semites. He dips liberally into the recent discussions of Babylonian civilizations and then runs rapidly through the middle ages.

The chapter on evolution is well worth reading and while there is very little that is new, there is much to think about and to admire, particularly in its presentation. One of the most interesting chapters is that on language and the primary sensations of hunger and love, and they are thoroughly taken up. The atavistic tendencies of the latter feeling are developed fully.

Chapter XII on the evolution of the brain, chapter XIII on the evolution of the senses and feelings, and chapter XIV on the instincts and emotions, make a unique series of heterogeneous facts brought into correlation by an ingenious method of arrangement.

Finally the author discusses the general problem of sociology in a general manner. He does not attempt a complete monograph of all of the various phases of the science of sociology but gives a number of scraps of interesting details.

This work is one that can be picked up in one's odd moments and from it can be gathered a rich store of useful information and pleasing fancy. We commend it most highly to our readers. JELLIFFE.

# News and Notes.

THE *Revista Freniatria Española* is a new journal devoted to psychiatry published in Spain at the beginning of this year.

THE ITALIAN SENATE has approved the application for the construction of a new building for the psychiatric clinic of the University of Pavia and has authorized the expenditure of $40,000 for the construction of the building and has also appropriated $12,500 for the care of patients.

THE National Asylum for Insanity of Rio de Janeiro, Dr. Juliano Moreira, professor of psychiatry and neurology on the Faculty of Bahia, and co-editor of the *Gaseta Medica* of Bahia, has been appointed Director of this large asylum. The institution is to be enlarged shortly by the addition of new buildings, the ground for which, adjoining the present property, has already been purchased. The erection of these new buildings will increase the capacity of the hospital to 1,000 beds instead of the present 900 beds. Funds for the erection of the buildings are available which will be sufficient to provide for a modern equipment of the new wards. Plans for the new epileptic ward are being prepared. It will be detached from the other buildings. The new children's ward was inaugurated with the name of *Pavilhas Bourneville*. Prof. Juliano Moreira, the new director, one of the most prominent alienists of South America, is author of several monographs in important points of psychiatry.

AT A RECENT CONFERENCE between Governor Odell and the New York State Commission in Lunacy plans were made for the erection of several new asylums for the insane and for the extensive improvement of the existing institutions, at a cost exceeding $1,000,000. The most important provision is for the erection in Washington county of a large hospital for insane, at a cost of $500,000. Accommodations will be provided for 1,200 patients, and arrangements will be made for large increases in a few years. It is also proposed to erect a psychopathic hospital in New York City for new cases picked off the streets. This institution will do the work of the Bellevue insane pavilion on a much larger scale. It will cost $250,000. Provision will be made for converting the Bedford House of Refuge for Women into an insane asylum, at an expense of $150,000. Tuberculosis pavilions for several state hospitals will cost $90,000. New boiler houses, kitchens and other departments at state hospitals will be provided for by an appropriation of $300,000.

DR. ARTHUR H. HARRINGTON, formerly superintendent of the Danvers, Mass., Hospital for the Insane, has taken charge of the New York Eye and Ear Infirmary, at New York.

A $16,000 wing is to be added to the County Insane Asylum, at Smith's Landing, N. J.

THE Clifton Springs, N. Y., Sanitarium management have found it necessary to erect a new bath house and otherwise enlarge and improve that institution in order to care for the increasing number of patients.

THE SUM OF $79,200 is asked for enlargements and improvements at the Middletown, N. Y., Homeopathic State Hospital. The sum of $25,000 is asked for solariums, $12,000 for a female hospital ward, $4,500 for additional heating apparatus, $3,500 for mechanical stokers, $15,000 for congregate dining room, $15,000 for another pavilion, etc.

IT IS STATED that a new sanitarium will be established at Amityville, N. Y. It is to be in charge of Mr. Gilbert P. Williams, former superintendent of the Brunswick Home.

DR. STOCKING, the assistant superintendent at Agnews, Col., State Hospital, has been appointed superintendent, vice Dr. J. A. Crane, resigned, while Dr. Marvin, who was second assistant at Agnews, has been appointed assistant.

THE STATE OF RHODE ISLAND is expending nearly $1,000,000 in the enlargement of the State Hospital for the Insane, at Cranston.

THE CONTRACT has been let for the erection of a $25,000 annex to Dr. Street's Sanitarium, at Vicksburg, Miss. It is to be completed by June 15.

THE PLANS have been completed for the erection of the new Resthaven Sanitarium, at Waukesha, Wis.

THE North Dakota State Hospital for the Insane, at Jamestown, is to be enlarged by the erection of a new hospital building and a cold storage building.

THE ERECTION of a $12,000 hospital at the County Asylum, at Nashville, Tenn., has been recommended by the board of commissioners.

THE NEW $75,000 building for the Waukesha Springs Sanitarium Co., at Waukesha, Wis., is to be completed about June.

F. D. BAYLESS is erecting a sanitarium at Prairie du Chien, Wis.

ST. AGNES' SANITARIUM, at Baltimore, Md., is to be extensively improved and enlarged.

DR. EDWIN S. VAIL is erecting a new $20,000 sanitarium at Thompsonville, Conn.

THE BATTLE CREEK SANITARIUM has opened a branch at Louisville, Ky.

DR. CHARLES LANG, of Meridian, N. Y., will erect a sanitarium at Pine Bluff, N. C.

THE POPE SANITARIUM, at Louisville, Ky., has been sold to the newly organized Pope Sanitarium Co., for $100,000.

THE LEGISLATURE has appropriated $80,000 for improvements at the State Hospital for the Insane, at Yankton, S. D.

PLANS have been filed for alteration to the old Amusement Hall on Blackwell's Island, New York, opposite the Metropolitan Hospital. After the alterations are completed the building will be used as a convalescent hospital. William Flanagan, Jr., of the Department of Charities, is the architect. The cost is placed at $10,000.

SHEPARD'S SANITARIUM, of Ohio, has been successfully conducted for fifty years without change of proprietor or management. With this excellent record Dr. William Shepard retired January 1, 1903, from active work and is now looking after his other varied interests. He will be succeeded by two corporations, The Shepard Sanitarium Company, for chronic and nervous diseases, and the McMillen Sanitarium Company, for mental diseases. More than a dozen well-known physicians of Central Ohio and vicinity are the stockholders in one or both of these sanitariums. A close relationship will be continued between them. Dr. W. E. Postle, of West Jefferson, O., will succeed Dr. Shepard, while Dr. Bishop McMillen will continue as superintendent of the sanitarium for mental diseases.

THE CONTRACTS were let early in February for the erection of eight new cottages for the Gallipolis, Ohio, State Hospital for Epileptics, to cost about $120,000.

EXTENSIVE IMPROVEMENTS are to be made this year at the Danvers, Mass., State Insane Hospital. A group of frame cottages, to accommodate one hundred patients, will be erected, as will also a dining room and kitchen and four wards and a heating plant.

THE NEW State Institute for Feeble-Minded, at Grafton, N. D., has been completed.

AMERICAN NEUROLOGICAL ASSOCIATION BANQUET.—At the last banquet of this Association held in Washington in May, some delightful fun was contributed by the members. G. P. W. contributed his annual song; J. C. cast an elaborate horoscope of many of the members, the dire forecast is not here reprinted lest the insurance rates in New York, Boston and Philadelphia should be disturbed. J. E. C. recited a fable and gave forth the following news item. Many others contributed to the jollity of the occasion.

IT IS RUMORED that Dr. M. P. is soon to publish a continuation of his delightful pipe-dream, otherwise known as the Genealogy and Development of the Misses Beauchamp. It will bear the imprint of the Society for Spookical Research and will have for title: The Pill Family Beauchamp. The dedication is to the late lamented Baron Munchausen. Mr. Charles Frohman has secured the dramatic rights.

A NEW AND UNIFORM edition of the works of Dr. J. C. is to appear in the fall. It will be done into English by the author himself and will contain no words of over twelve syllables. Since the apearance of Dr. C.'s books, Messrs. Flunk and Waxemall have been enabled to add over seven thousand new words to their peerless Standard Dictionary.

DR. P. C. K. denies the rumor that he is writing a book on Sex. Perv. He admits however that his extensive reading on this subject has for its object a short story dealing with literary and artistic circles, soon to appear in the Smart Set.

DR. W. G. S. is spending his leisure moments in the preparation of a monograph entitled: A Description of Two Newly Discovered Nerve Fibers in the Genito-Spinal Tract of the Common or Garden Flea.

IT IS RUMORED that Dr. H. P., of Salem, Mass., has another nervous patient. A fresh article may therefore be confidently expected from the prolific pens of Drs. W. and P., who will doubtless see the case in consultation. The healthy young women of the Normal School of Gymnastics will probably lend their physical charms to the construction of the paper, as usual. (G. E. C.)

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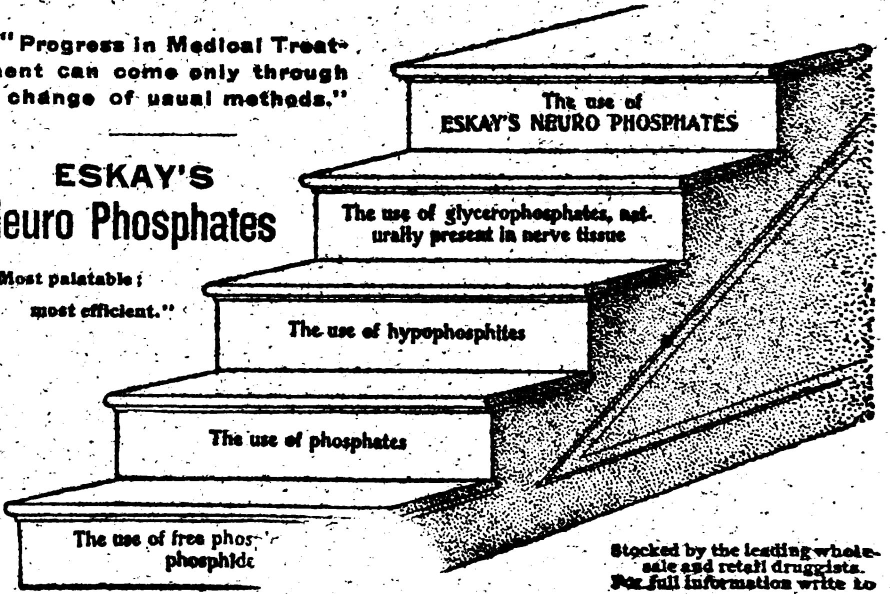
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Phil 19.2

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The Official Organ of

The American Neurological Association

The New York Neurological Society

The Philadelphia Neurological Society and

The Chicago Neurological Society

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FOREIGN AND DOMESTIC AGENTS
S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. [illegible] & Co., 6 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

Printed by the Alliance Press Company, 23 W. 16th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.50 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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## Original Articles.

# A CASE OF PROGRESSIVELY DEVELOPING HEMIPLEGIA, LATER BECOMING TRIPLEGIA, RESULTING FROM PRIMARY DEGENERATION OF THE PYRAMIDAL TRACTS.[1]

By Charles K. Mills, M.D.,
CLINICAL PROFESSOR OF NERVOUS DISEASES IN THE UNIVERSITY OF PENNSYLVANIA; NEUROLOGIST TO THE PHILADELPHIA HOSPITAL,
AND
William G. Spiller, M.D.,
ASSISTANT CLINICAL PROFESSOR OF NERVOUS DISEASES, AND ASSISTANT PROFESSOR OF NEUROPATHOLOGY IN THE UNIVERSITY OF PENNSYLVANIA; NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

In this contribution the clinical and pathological details of a case of progressively developing hemiplegia, which later became triplegia, due to primary degeneration of the pyramidal tracts, will be presented, with a review of the literature of similar clinical cases. A summary of the literature of primary bilateral sclerosis will also be given.

J. S., white, was admitted to the Philadelphia Hospital August 9, 1897, where he died January 14, 1903. He was fifty-four years old when admitted. With the exception of a short time during which he was treated in the Men's Medical Ward for diarrhea or dysentery, he was during this period of six years a patient in the Men's Nervous Ward.

Omitting some unessential points, his history as traced in the hospital records is as follows: The family history obtained has no etiological value. He denied venereal disease. He was a heavy drinker and also used tobacco. He had had rheumatism six years before admission to the hospital. The earliest note referring to any nervous symptoms was made August 9, 1897, when a statement was recorded that two years before admission he had been paralyzed on the right side. It is probable that at this time (during 1890-1895) his loss of power first began to show itself.

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[1] Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

From the William Pepper Clinical Laboratory, Phœbe A. Hearst Foundation.

On September 28, 1897, the record stated that right hemiplegia (more correctly hemiparesis) was present, that the biceps-jerk and knee-jerk were increased, and that the plantar reflex was absent. Ankle clonus also was absent.

On September 24, 1899, it was noted that he could use his right leg for various willed movements, but that it showed loss of power as compared with the left; also that there was some loss of power in the right arm as compared with the left. His grip measured by the dynamometer indicated fifty-five for the right and seventy-five for the left. Speech was a little thick. The knee-jerk on the right was much exaggerated, and the quadriceps-jerk and gastrocnemius-jerk were also increased. Both ankle-clonus and patellar clonus of moderate degree were present on the right. No note was made at this time as to the Babinski phenomenon. Biceps-jerk, triceps-jerk and all muscle jerks in the right upper extremity were increased. The tendon and muscle responses on the left were all present but not exaggerated as on the right, and patellar clonus and ankle clonus were absent on the left. Sensation for touch and pain was retained all over the body, and the stereognostic perception was undisturbed. The irides reacted to light and in accommodation, and other examinations for nervous phenomena gave negative results.

On May 17, 1901, the patient complained that the right leg was weaker than it had been one year previous. The reflexes and other symptoms were much as before recorded.

Although the case was seen from time to time no further notes as to his nervous symptoms were made until December 29, 1902, at which time it was recorded that the movements of the face and tongue were normal; the upper limbs could be moved voluntarily and fully in all parts; the grasp of the right hand was feeble, of the left normal; voluntary power in the right lower limb was very slight; in the left lower limb it was a little better than in the right but much impaired. The right upper limb and both lower limbs were spastic, but contractures and wasting were not present. The biceps, triceps and wrist reflexes were much exaggerated in the right upper extremity, and in the left they were a little more prompt than normal. Patellar and Achilles reflexes were exaggerated on both sides, but more so on the right; patellar clonus was present on the right but not on the left; ankle clonus was elicited on each side. The Babinski reflex was present but not very prominent on the right; it was absent on the left. Sensation to touch and pain was everywhere normal.

This man suffered at times from severe intestinal and cardiovascular symptoms. On August 6, 1897, he began to have severe pain in the abdomen with diarrhea, with numerous stools accompanied by straining and pain. He was treated for these symptoms in the medical wards. The heart sounds at this time were feeble and accompanied by a low systolic murmur at the right base. The sounds were feeble on the left also, and were best at the ensiform cartilage. On December 11, 1898, the records show that he had a soft blowing systolic murmur at the second aortic interspace. May 31, 1899, the patient complained of pain in the chest most marked over the precordia. A soft double systolic murmur was heard above the second cartilage. The heart's action was weak and irregular. A few unimportant notes, chiefly negative, were made in September, 1899. The presence of the cardiac murmurs above described was reiterated. On January 17, 1902, the patient complained of constant and severe pain over the precordial region; the heart sounds were very feeble. The pulse on the left side was imperceptible, owing to the atheromatous condition of the arteries and to the cardiac weakness. The pulse on the right side was perceptible but weak, and the radial arteries on both sides were hard and rigid. The patient suffered at this time and later from cardiac weakness and anginal pains for which he was treated with various remedies.

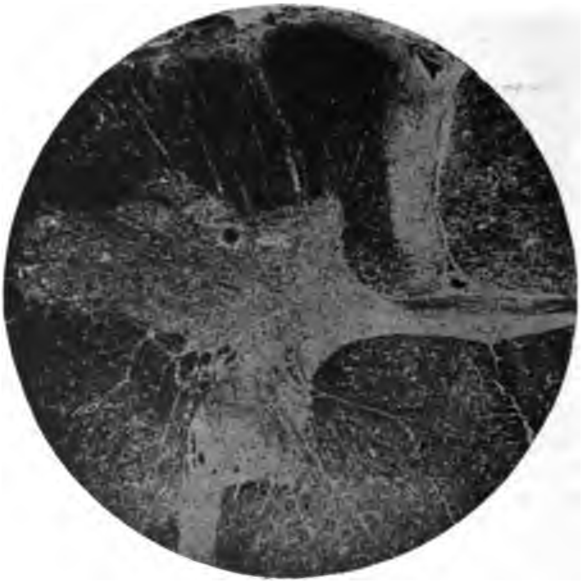
About January 1, 1903, he began to show signs of great weakness and rapid failure. He had at times attacks of vomiting. He lost flesh, his pulse became weak and intermittent, and the right lung became edematous. He died January 14, 1903, his death being preceded by a slight convulsion.

The necropsy showed a serious pathological condition of the vessels and many of the viscera: chronic pleurisy and tuberculosis with cavity formation; endocarditis, contracted kidneys and chronic marked atheroma. The heart muscles were soft and pale, the valves distorted, thickened and calcareous, the aortic extensively atheromatous. Both kidneys were very firm, dark in color, with granular surface and irregular cortex. The stomach was decidedly contracted and the seat of chronic catarrh. Beginning at the junction of the transverse and descending aorta was a unilateral dilatation of this vessel, which increased in diameter to about 4 cm. This structure was atheromatous and contained a flattened clot. The brain and cord were removed for microscopical examination, of which the following is a record.

The degeneration in the lumbar region was very distinct by the Weigert hematoxylin stain in the crossed pyramidal tracts, and more intense in the right than in the left. The direct pyramidal tracts by this stain did not appear degenerated at this level of the cord. The nerve cells of the anterior horns were not distinctly altered, they were numerous and some were much pigmented, which, considering the man's age, was not remarkable. The chromophilic elements were well formed and the nuclei central. It may be said that the nerve cells of the anterior horns of the lumbar region had the usual appearance in persons of the same age. The anterior roots on each side in the lumbar region were normal.

At the eighth cervical segment the degeneration of the right crossed and left direct pyramidal tracts was very distinct by the Weigert hematoxylin stain. The left crossed and right direct pyramidal tracts were much less degenerated. The nerve cells of the anterior horns of this region may be said to have been normal, although in examining a number of sections a few cells were found that were not normal: the nerve cells in general were remarkable for their excellent state of preservation.

Sections from the lower cervical region stained by the Marchi method showed no recent degeneration in the right crossed and left direct pyramidal tracts. The left crossed and the right direct pyramidal tracts contained numerous small black dots, and those of the crossed tract were much smaller and less numerous than those of the direct tract. At the outer border of the right direct



Long-standing degeneration of the left direct pyramidal tract, recent degeneration of the right direct and of the left crossed pyramidal tracts. The dots in the crossed tract are not so large as those in the direct tract (Method of Marchi).

pyramidal tract the dots were smaller than in the median and inner portions of this tract.

The anterior roots in the lower cervical region were normal. There was no distinct meningitis; here and there a slight accumulation of round cells was found in the pia.

Both pyramidal tracts were much degenerated in the oblongata and pons, but the left tract was much more degenerated than the right. The right pyramidal tract was much degenerated in the oblongata and pons by the Marchi method, while the left showed no recent degeneration by this method. About the middle of the pons, the left pyramidal tract contained a large proportion of normal nerve fibers, but a part of it was much degenerated. No degeneration could be detected in the foot of the right or of the left cerebral peduncle by the Weigert hematoxylin method, but degeneration by the Marchi method could be found in the middle portion of the foot of the right cerebral peduncle and also where the foot of the right cerebral peduncle becomes the lower part of the posterior limb of the internal capsule; the degeneration however was slight. No degeneration could be seen by the Marchi method in the foot of the left cerebral peduncle.

Sections of the left paracentral lobule contained Betz cells that appeared normal. They were not numerous, but this does not mean that there had been a loss of the cells, as the number of these cells seems to vary greatly in different cases.

Cellular infiltration of moderate intensity was found in the pia about the cerebral peduncles and pons, but it seems hardly probable that this could explain the long-standing degeneration of the pyramidal tract from the left cerebral hemisphere, and for this reason we have spoken of the degeneration of the pyramidal tracts as primary. No focal lesion could be detected anywhere.

Summarized, this case was one in which hemiplegia gradually developed on the right side, the lower extremity being more markedly and probably earlier affected than the upper, the case therefore at first belonging to the clinical type of unilateral progressive ascending paralysis. After several years the left lower extremity also became paralyzed, but not to the same extent as the right. The reflexes were all markedly exaggerated, the Babinski response being present. Sensory symptoms were absent. Microscopical examination showed intense and long-standing degeneration of the right crossed and the left direct pyramidal tracts, the degeneration extending into the pons but not into the left cerebral peduncle; also comparatively recent degeneration of the left crossed and the right direct pyramidal tracts, traced by the method of Marchi into the lower part of the right internal capsule. No lesions, degenerative or focal, were found elsewhere in the brain or spinal cord; the case, therefore, was one of primary degeneration of the motor tracts, much greater and older in the right crossed and left direct pyramidal tracts. The case may be regarded as a corroboration of the clinical type described by Mills in the JOURNAL OF NERVOUS AND MENTAL DISEASE for April, 1900.

In the JOURNAL OF NERVOUS AND MENTAL DISEASE for April, 1900, in a paper entitled "A Case of Unilateral Progressive Ascending Paralysis, Probably Representing a New Form of Degenerative Disease," which was presented to the Philadelphia Neurological Society on December 18, 1899, an interesting case is detailed, reference being made to another somewhat similar case previously observed. The patient, a man fifty-two years old, about two years before coming under observation began to show signs of weakness in the right lower extremity, as indicated by his method of walking. The paresis came on slowly and insidiously. Weakness in the arm appeared for the first time eighteen months after the weakness was noticed in the lower extremity. This weakness soon became more and more evident, and was accompanied by the tendency to carry the arm raised against the body and flexed at the elbow. When first examined the paresis in the upper extremity, although easily determined, had not nearly reached the degree of impairment observed in the leg. The right side of the face was also slightly but undoubtedly paretic. The patient had had at one period some hyperesthesia in the right lumbar region and in the lower extremity, and later in the right upper extremity. Herpes appeared in the lumbar and lumbosacral regions, lasting for a short time. Wasting was distinct in the right lower extremity, the measurements showing a difference of one and a half inches for the thigh, and five eighths of an inch for the calves. The various movements of the right leg were distinctly weaker than those of the left, but were nowhere absolutely abolished. Similarly all the movements of the right arm were distinctly impaired, but were nowhere absolutely lost. The dynamometer showed 180 for the right and 160 for the left. Faradic contractility was retained. The affected limbs were not spastic nor contractured. Careful examination showed retention of all forms of sensation. The tendon and muscle phenomena on the right side were all somewhat exaggerated. Knee-jerk was plus on the left side, but was considerably more exaggerated on the right. Patellar clonus was present on the right but not on the left, and the right side showed a slight ankle clonus, which was absent on the left. The plantar reflex was normal on the left; but on the right, while the Babinski reflex was not present, the normal response was distinctly less marked than on the left. It might be described as between normal plantar flexion of the toes and the dorsal flexion of the Babinski reflex.

This case has recently been made the subject of reinvestigation by Dr. Hugh T. Patrick, who inclines to the opinion that the case is one of paralysis agitans without tremor, the disease up to the present time having attacked only one side of the body. His report of the case was presented at the meeting of the American Neurological Association, and will be found in its Proceedings, with the discussion of the case by Dr. Mills. At the time when the patient was examined by Dr. Mills, among the reasons for believing that the case was not paralysis agitans were the absence of spontaneous tremor; the presence of markedly exaggerated reflexes on the affected side, even including ankle clonus and patellar clonus; the existence of decided wasting on one side, especially of the lower extremity, and the absence of the facies of paralysis agitans and of a fixed position of the body and head.

According to Sir William Gowers, both primary lateral sclerosis and paralysis agitans are probably abiotrophies (*Lancet,* April 12, 1902), and it may be that in the case under consideration the two abiotic diseases are conjoined.

In the same paper in which this case is detailed a second case is reported. This patient was under the care of Dr. Mills seventeen or eighteen years ago, and had previously been a patient of the late Dr. E. C. Seguin of New York, who believed that there was a cerebral lesion causing changes in the crossed pyramidal fasciculus analogous to lateral sclerosis. The view taken by Mills was that the changes in the crossed pyramidal tract were of the nature of a lateral sclerosis, but were primary and not secondary to any cerebral lesion. The patient was a woman forty-three years old, who first noticed weakness in her leg while pregnant with her last child, three years before coming under observation. The left arm became paretic a few months after the left leg. The reflexes were much increased on the affected side. The patient remained under observation for many months, the paresis of the leg and arm slowly increasing. Neither arm nor leg was contractured, and sensibility was preserved. The patient complained at times of pains like neuralgias in the limbs, and of nervous twitching in the leg and arm. This patient was alive three or four years ago, and had become entirely unable to walk, but just how she was affected was not learned.

In the paper reporting these cases the differential diagnosis from an unusual form of unilateral disseminated sclerosis, from unilateral amyotrophic sclerosis, from a progressive hemiplegia due to slowly increasing focal cerebral lesion involving the motor subcortex or the internal capsule, from a degenerative motor neuritis, and from a functional hemiparesis was discussed. Reasons were given why the cases did not seem to fit in exactly with any one of these diagnoses.

In addition, as has been indicated above, the diagnosis must be made from unilateral paralysis agitans. Syphilitic hemiplegia later becoming a triplegia might also have some points in common, but could usually be separated by the presence of such concomitant syphilitic affections as paralyses of the cranial nerves.

In the *Philadelphia Medical Journal*, Feb. 9, 1901, Spiller has recorded the case of a man, forty-one years old, who began, four years before coming under observation, to feel weak in the left lower limb while walking. The weakness increased, and in about a year the left upper extremity also showed signs of feebleness. He was never unconscious and never had headache or vertigo or other signs of focal intracranial disease. When the case was recorded the movements of the left lower limb were spastic, but not ataxic; the toes of the left foot were scraped along the ground and the foot turned inward. Both knee-jerks were prominent, but the left much more so than the right. Ankle clonus was obtained on the left but not on the right, and the Babinski reflex was distinct on the left, but uncertain on the right. Sensation was normal. The resistance to passive movement and the grasp of the hand were decidedly weak. The left upper limb was weaker than the right; it was also spastic, but the weakness and spasticity were less than in the lower extremity. The left upper limb was held slightly flexed at the elbow and against the body, but no contractures were present anywhere. Speech was normal, but the mouth could not be drawn up as well on the left side as on the right. The tongue went slightly to the left on protrusion. Ophthalmoscopic examination by Dr. H. F. Hansell showed white atrophy of the left optic nerve. After giving the details of his case, Spiller discusses its probable diagnosis, and concludes that the cases reported by Mills and by him have the symptoms which one should expect from unilateral lateral sclerosis, but he also called attention to the fact that other lesions might cause the same symptoms. The wasting is like that occurring in hemiplegia.

Potts (JOURNAL OF NERVOUS AND MENTAL DISEASE, Oct. 1901), under the title, "A Case of Progressive Unilateral Ascending Paralysis Probably Due to Multiple Sclerosis," has reported a case somewhat similar to the case described by the authors of this paper. The patient, a youth of nineteen, when about fifteen years old began to drag his right foot, this foot in walking showing a tendency to cross over the other. Two years after the impairment of power, diplopia appeared and remained for five months. It was discovered at about this time that the right leg was smaller than the left. Weakness in the right arm was first noticed about three and a half years after its appearance in the right leg. Examination showed that when the patient walked the right leg was rigid and the toe dragged. All movements of the leg were preserved but impaired. The right arm was distinctly weaker than the left, but was not spastic. Both the arm and leg were decidedly atrophied, as shown by careful measurements. The adductor pollicis was not so large or firm as the left. The reflexes were prompt everywhere but exaggerated on the right, the Babinski response being present. The right side of the face showed slight paresis with some tremor on voluntary effort. Paresis of the muscles of the right side of the throat was present. Nystagmus was present especially when the eyes were turned to the right. There was paresis of the left inferior rectus, and paleness of the temporal halves of the discs. All forms of sensation, including stereognosis, were normal. A slight Romberg symptom and tendency to incoördination were sometimes shown. The right hand and foot were colder to the touch than the left. Potts, while recognizing the resemblance between his case and the cases reported by the writers, holds that his case is probably one of disseminated sclerosis, and suggests that our cases might also be classed in this category, a view which we cannot accept. This diagnosis in Pott's case may be correct, although it is also probable in this and similar cases of disseminated sclerosis that the degeneration first and for a long time is chiefly confined to the pyramidal tract on one side.

Gowers, in his paper on "Abiotrophy" (*Lancet,* April 12, 1902), says truly "that the symptoms of systematic degeneration are not alone ground for the diagnosis of insular sclerosis, nor is the additional presence of nystagmus. Nystagmus on movement of the eyes is met with in many spinal degenerations of pure systemic type."

In his paper on "Chronic Progressive Hemiplegia," read at the present meeting of the American Neurological Association, Patrick describes a case which he regards as clearly indicating the progressively developing hemiplegia due to degeneration of the pyramidal tract of one side. During a recent visit to Chicago one of us (Dr. Mills) had the opportunity, through the courtesy of Dr. Patrick, of seeing this case, that of a young woman, eighteen years old, whose motor disability appeared about four years previously. The essential features of this case are the unilateral and progressive character of the paralysis which probably began first in the lower extremity, the exaggeration of the reflexes on the affected side, the absence of all forms of sensory disorder and of visceral symptoms, and the moderate wasting of the limbs and face. The reader is referred to Dr. Patrick's paper for fuller particulars of this case.

Another case of unilateral progressive ascending paralysis has been in our care at the University Hospital.

From a study of the cases presented in this paper, and in the light of the pathological findings in the case with autopsy, it may be concluded that there is a form of progressively developing hemiplegia, usually of ascending type, sometimes becoming triplegia or even quadriplegia, due to a progressive primary degeneration of the pyramidal tracts, which begins on one side and may extend to the other. This clinical picture may be produced by other lesions, but we believe that we have established the fact that primary progressive degeneration of one pyramidal tract occasionally occurs. This is a unilateral lateral sclerosis, and in this connection it is interesting to devote some space to the subject of bilateral lateral sclerosis.

In a recent address in London, Erb[2] spoke of the spastic spinal paralysis first described by him in 1875. He believes that sufficient cases have been published to show that primary bilateral sclerosis exists. The following cases he gives as indisputable, and capable of standing any criticism.

Case 1. Minkowski.[3] Clinically the picture was one of spastic spinal paralysis. Anatomically there was a nearly pure sclerosis of the crossed pyramidal tracts, with slight changes in the direct cerebellar tracts.

Case 2. Von Strümpell.[4] The patient was a man, aged sixty-three years, who had a brother suffering from the same disease. Clinically the case presented the typical picture of spastic spinal paralysis for at least twenty years. Anatomically there was a typical degeneration of the pyramidal tracts from the lumbar to the cervical region, and in addition a slight degeneration of the direct cerebellar tracts and a still more trifling degeneration of the tracts of Goll in the upper part of the spinal cord. Strümpell would, on this account, reckon the affection among the combined system diseases, but the degeneration of the pyramidal tracts was certainly the most essential lesion, and is to be regarded as primary.

Case 3. Dejerine and Sottas.[5] Clinically this was a pure case of spastic spinal paralysis of twenty-five years' duration, death occurring from pneumonia in the sixty-sixth year. Anatomically there was marked sclerosis of the pyramidal tracts from the lumbar to the cervical cord, with slight partial sclerosis of Goll's tracts in the cervical region.

Case 4. Donaggio.[6] The patient was a man aged sixty-one years. Clinically there was for two and a quarter years the typical picture of pure spastic spinal paralysis; death resulted from pneumonia. Anatomically there was pure, exclusively primary degeneration of the pyramidal tracts from the lumbar to the cervical region (a quite typical case).

Case 5. Friedmann.[7] The patient was a male, aged fifty-two years. Clinically for two years the picture was one of a spastic spinal paralysis (trace of disturbed sensation being present?). The patient suffered from apoplexy, death resulting from pneumonia. Anatomically there was classical primary degeneration of the pyramidal tracts only, there being a trace of degeneration in the direct cerebellar tracts as well as endarteritis obliterans of the basilar artery.

Case 6. Von Strümpel.[8] Clinically a picture of spastic spinal paralysis was present, death occurring after the disease had lasted for thirty-five years. Anatomically there was quite typical moderate degeneration of the pyramidal tracts from the lumbar cord to the region of the pyramids. The direct cerebellar tracts were scarcely affected and the tracts of Goll very slightly in the upper cervical region. The anterior columns and brain were entirely free (belongs to the hereditary form).

Cases 7 and 8. Bischoff.[9] The patients were two brothers who, from their eighth and tenth years respectively, suffered from spastic rigidity of the limbs, ascending slowly from the legs to the head. Intellectually development was poor, but otherwise clinically the typical picture was one of spastic spinal paralysis. Death took place from phthisis after about twenty years' duration. Anatomically there was a typical degeneration of the pyramidal tracts, extending upwards beyond the oblongata, but not farther. The direct cerebellar tracts and Gowers' bundles were all but free. Goll's tracts were extremely affected. In the gray matter of the anterior columns there was atrophy of the ganglion cells (evidently occurring towards the end of life). The author calls the change in the spinal cord primary tract degeneration (quite analogous to the condition found by Strümpell), and holds that thereby the existence of an infantile form of hereditary spastic spinal paralysis has been proved.

Case 9. Ida Democh.[10] The case is clinically and anatomically somewhat complicated; clinically there was a typical picture of spastic spinal paralysis, combined with signs of chronic alcoholism, pains, tremors, etc. Anatomically there were primary degeneration of the tracts of Goll and congenital hydromyelia in the lumbar and dorsal cord; the direct cerebellar tracts were free; nevertheless, as proved by the author, this can be termed a case of primary degeneration of the pyramidal tracts in spastic spinal paralysis.

Case 10. A case clinically typical of spastic spinal paralysis described by Kühn, in which von Strümpell made the microscopical examination of the spinal cord. He found an essentially characteristic primary degeneration of the lateral columns.

Erb regarded the sclerosis of the direct cerebellar tracts and the slight degeneration of the columns of Goll in some of these cases as of secondary importance.

He thinks that the case described by Strümpell[11] in 1894 as a primary isolated system degeneration of both pyramidal tracts, showing only slight transitions to amyotrophic lateral sclerosis, might come under this head.

To this record we would add the case reported by us in this paper, and also the case reported by one of us (Spiller[12]) in which there was primary degeneration of the pyramidal tracts with comparatively slight alteration of the nerve cells of the anterior horns of the spinal cord.

Note—Since this paper was read, another communication by Erb on spastic spinal paralysis has been published in the *Deutsche Zeitschrift für Nervenheilkunde,* Vol. 23, Nos. 5 and 6. He refers to the ten cases mentioned in his previous paper, and includes the well known case of Morgan and Dreschfeld.

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[2] Erb, Lancet, 1902, p. 970.

[3] Minkowski, Deutsches Archiv f. klin. Med., Vol. 34, 1884.

[4] Von Strümpell, Archiv f. Psych., 17, 1886.

[5] Dejerine and Sottas, Archives de Physiologie Normale et Pathologique, 1896, p. 630.

[6] Donaggio, Rivista sperimentale di freniatria, Vol. 23, 1897.

[7] Friedmann, Deutsche Zeitschr. f. Nervenheilk., Vol. 16, 1899.

[8] Von Strümpell, Neur. Centralbl., 1901, p. 530.

[9] Bischoff, Jahrbücher f. Psych. und Neur., Vol. 22, 1902.

[10] Ida Democh, Archiv. f. Psych., Vol. 33, 1900.

[11] Von Strümpell, Deut. Zeitschr. f. Nerv., Vol. 5, p. 225.

[12] Spiller, Journal of Nervous and Mental Disease, May, 1902.

# THE RESULTS OF SURGICAL TREATMENT OF BRAIN TUMORS.

By M. Allen Starr, M.D., LL.D.,
of New York.

In order to estimate accurately the prospect of success in any surgical operation, it is wise to consider the results which have been attained in a large number of cases. And while, too great importance should never be attached to statistics, yet on the other hand, they possess a certain value and are never without an element of interest. In 1893, I was able to collect seventy cases of brain tumor in which an operation had been undertaken,[1] and in 1896 and 1897, I analyzed the results in two hundred and twenty cases in which a surgical operation had been attempted.[2] As a knowledge of the possibility of operation has become more widespread, and as the technic of brain surgery has become more perfect, reports of such operations have been more frequent, and it has been possible to collect 365 cases up to January 1, 1903.[3] In the appendix to this article the cases which have been published since January 1, 1896, are tabulated, all those prior to that time being referred to either in *Brain Surgery,* or in the *Medical Record* for February 1, 1896, or in the article in the *Montreal Medical Journal* for November, 1897.

TABLE OF RESULTS OF OPERATION FOR BRAIN TUMOR.

| | | *Cerebral.* | *Cerebellar.* |
|---|---|---|---|
| Total number of cases operated upon | 365 | 315 | 50 |
| Cases in which the tumor was not found | 111 | 91 | 20 |
| Cases in which the tumor was found but not removed | 27 | 21 | 6 |
| Cases in which the tumor was removed and the patient died | 59 | 51 | 8 |
| Cases in which the tumor was removed and the patient recovered | 168 | 152 | 16 |

There are two elements which render the chances of success in the operation for brain tumors better at present than formerly.

(1) The accuracy of diagnosis and the accuracy of localization

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[1] Brain Surgery, Wm. Wood & Co., 1893.

[2] New York Medical Record, February, 1896. Montreal Medical Journal, November, 1897.

[3] These cases have been gathered from the literature for me by Dr. O. Hensel.

of tumor are today more easily possible than before. Given a history of a slowly progressing disease with symptoms of headache, vomiting, vertigo, sensations of cerebral discomfort, progressive emaciation and feebleness, and a gradual loss of sight due to a choked disc, and the only probable hypothesis is the existence of a brain tumor. No other disease can produce this particular combination of symptoms. Hence the diagnosis of the nature of the disease is not difficult.

The possibility of locating tumors has also become more definite within the last twenty years. This diagnosis is wholly by direct observation of local symptoms such as (a) mental symptoms, (b) motor or sensory aphasia, (c) local spasm and monoplegia, (d) hemianopsia, (e) cerebellar staggering.

The diagnosis is also one to be reached in part by elimination, for the absence of any one of these localizing symptoms just named, when the disease is clearly a brain tumor, makes it evident that the tumor is inaccessible. It is therefore possible at the present time to determine with considerable accuracy, *first,* that a tumor is present, and, *second,* that the surgeon can reach it.

The chance of success has also been materially increased by the newer surgical methods of access to the brain. It is true that successes had been obtained by the use of the ordinary trephine, the opening being enlarged by a rongeur. It is also true that the method of opening the skull by a horseshoe-shaped incision through scalp, periosteum and bone, by means of a gouge and chisel, has been attended by greater success. This method was a distinct improvement upon the method by the use of the trephine. But during the past three years these two methods have been superseded by the use of the electrical saws. In the larger hospitals it is perfectly possible to obtain a powerful electrical engine connected with a flexible tube, on the end of which revolving spools can be inserted. The method at present used by McCosh at the Presbyterian Hospital seems to me the best method as yet devised for entering the skull. A small bulb of the shape of a pear, about one-quarter of an inch in diameter, is attached to the electrical engine, and with this bulb four or five small holes are bored through the skull. The cone-shape of the bulb allows the point to penetrate through the skull as it is revolved, and yet prevents the shaft from entering the skull or penetrating the dura. It requires but a few seconds with the powerful revolutions of this bulb, that are obtained by the machine, to bore one of these holes. The number of holes and their position upon the skull are determined entirely by the area that it is desired to expose. If four holes are bored they are from one and a half to four inches apart. When these holes have been made the bulb is removed from the machine and there is substituted a small circular saw and the bone between these holes is quickly sawed through by it. A small projecting and protecting flange is screwed to the side of the circular saw so that the blade of the saw is exposed only for the depth of the skull as measured through the holes, and in this way the danger of sawing through the dura is wholly obviated. It requires hardly a minute when the saw is properly adjusted to saw through the skull from one hole to the next. In this manner the skull can be opened in from six to eight minutes after the original incision has been made through the scalp. This method offers great advantages in the rapidity with which access can be had to the brain.

Experience has taught that it is a great advantage in the extraction of tumors to have plenty of space, and it has also taught that these large bony flaps can be replaced and will unite without any danger. The bony flap is turned downward, as a rule, the lower edge of the flap not being sawn entirely through, but cracked. It is left attached to the periosteum and skin. The incision in the scalp will be determined in its shape by the incision which it is proposed to make in the bone. This may be rectangular or polygonal. Much time is saved in the operation by making the bony flap of large size, as in former operations it was often necessary to delay for the enlargement of the bony opening after the tumor had been found. By the use of this method, I have frequently seen the brain exposed within ten minutes of the time of the beginning of the operation; while in prior methods from eighteen to twenty-five minutes were often expended in this preliminary stage.

Another factor in success in these operations is the fact that we have come to the conclusion that extensive cutting of the brain and exsection of parts of the brain are not necessarily attended by danger, provided the hemorrhage can be arrested. I have seen large areas, two to three inches in diameter, cut out of the brain, to a depth of two to three inches, and I have seen the patients recover. While the difficulties attendant upon the extraction of a deep tumor are many, and while the chances of subsequent recovery, first, from the operation, and, second, from the symptoms, are much less in such deep tumors, yet it must be conceded that a tumor lying one or two inches below the surface may be reached.

For these reasons it seems to me probable that the statistics of the next ten years will show a very much greater percentage of successes than do the statistics up to the present time.

The operation for cerebellar tumors appears to me to be of great difficulty and danger. But one-tenth of each hemisphere of the cerebellum can be reached by the surgeon through the occipital fossa. This I have determined by measurements of the surface many times in the cadaver. The surface which is accessible is one which is very rarely the seat of a cerebellar tumor. Cerebellar tumors lie either in the middle lobe of the cerebellum directly beneath the tentorium, a point which cannot be reached from the lower surface of the cerebellum, or else they lie in the sulci between the cerebellum and the pons and medulla oblongata, upon the base of the brain, a point which is equally inaccessible. For this reason failures are numerous in operations for cerebellar tumor, and after a considerable experience (I have had nine such cases operated upon without any success) it seems to me futile to attempt this operation.

The causes of failure are: First, mistakes in the diagnosis of the location of the tumor. In a certain percentage of tumors these mistakes are inevitable. Certain local symptoms of tumor which appear to point to a clear localization do occur in some cases when the tumor is at a considerable distance from the point where it is supposed to lie. The local symptoms are therefore not infallible in their indication.

Secondly, some tumors, even when accessible, cannot be removed. They are infiltrating gliomata without any boundary and with a vascular, softened tissue about them which prevents enucleation, and this variety of tumor is particularly liable to return even if removed in part.

Thirdly, the dangers of hemorrhage and the dangers of meningitis are less dreaded at present than formerly, proper asepsis preventing the latter complication in all cases.

### APPENDIX BY DR. O. HENSEL.

### VARIETY OF TUMOR REMOVED AND RESULTS.

### 1893-1903.

| | *Recovered.* | *Died.* |
|---|---|---|
| Sarcoma | 34 | 13 |
| Melanosarcoma | 1 | — |
| Cystosarcoma | 1 | — |
| Gliosarcoma | 5 | — |
| Glioma | 8 | 5 |
| Neuroglioma | 2 | — |
| Perithelioma | 1 | — |
| Endothelioma | 3 | 3 |
| Carcinoma | 1 | — |
| Hydatid Cyst | 4 | 1 |
| Cyst | 22 | 7 |
| Tubercle | 11 | 4 |
| Gumma | 5 | 2 |
| Fibroma | 1 | 2 |
| Angioma | 4 | — |
| Osteoma | 1 | — |
| Not stated | 16 | 9 |
| | 120 | 46 |

### TUMOR TREPHINED FOR BUT NOT FOUND.

*Central Region.*—1-2. Bramwell, *Boston Med. Journ.*, Dec., 22, 1900, 2 cases, glioma right motor region, no tumor found, death in 9 months.—3. Hoppe, *Journ. of Amer. Med. Assoc.*, Feb. 2, 1901, Tubercle in lentiform nucleus of right side, extending to within ½ inch of cortex in ascending frontal and parietal regions and inward and downward to internal capsule. Death in 6 hours.—4. Hoppe, *ibid.*, Gliosarcoma, extending 2 inches from tip of 1st frontal convolution, back along longitudinal fissure to 2 inches posterior to fissure of Rolando. Death on same day.—5. Hoppe, *ibid.*, Multiple cysts. Trephined at lower extremity of fissure of Rolando. Death from cerebral hemorrhage.—6. Schulze, *Deutsche Zeitschrift f. Nervenheilk.*, 1896. Sarcoma in left facial center, death.—7. Clarke, *Glasgow Med. Journ.*, 48, p. 13. Motor area trephined, not found, death, no autopsy.

*Frontal Region.*—8. Hitzig, *Therap. Wochenschrift*, 1896. Glioma, right frontal lobe, death soon after operation.—9. Zenner, *Transactions of American Neurological Association*, May, 1897. Subcortical growth in prerolandic gyrus.—10. Stochow, *Inaugural Dissert.*, Erlangen, 1896. Tumor, left frontal lobe on autopsy.—11. Hahn and Ruheman, *Deutsche med. Wochenschrift*, 1896. Trephined in frontal region, not found. Was in 2d and 3d frontal convolution. Improvement.—12. Clarke, *British Med. Journ.*, Oct. 13, 1900. Glioma, left frontal lobe, not found, death next day.—13. Armitage, *Lancet*, Nov. 26, 1898. Cortical growth, superior and middle frontal convolution.—14. Gordinier, *American Journ. of Med. Sciences*, May, 1899. Glioma, second left frontal convolution.

*Parietal Region.* — 15. Williamson, *British Med. Journ.*, July 6, 1901. Sarcoma at right parieto-occipital fissure, death in 1 month.—16. Rossolimo, *Arch. f. Psych.*, xxix. Cavernous angioma of left paracentr. lobe, death in 15 hours of hemorrhage.—17. Lambotte, *Annals de la Soc. Belg. de Chir.*, 1896. Trephined in occipital region. Was in angular gyrus. Death.

*Temporal Region.*—18. Gessler and Bayha, *Würtemb. ärst. Correspbl.*, 1897. Tumor in temporal lobe, death in 6 days. —19. Starr and Weir, *Medical News*, Aug. 7, 1897. Glioma.—20. *Transactions of the American Neurological Association*, May 5, 1897. Sarcoma in left temporal lobe.—21. Starr, *British Med. Journ.*, Oct. 16, 1897. Glioma, trephined for in left 3d frontal convolution, death.

*Occipital Lobe.*—22. Barette, *Soc. de Chirurg. de Paris*, Nov. 11, 1897. Neuroglioma of occipital lobe, compressing longitudinal sinus and causing a swelling in the frontal region, which could not be removed on account of hemorrhage.

*Deeper Parts of Brain.*—23. Mingazzini, *Deutsche Zeitschrift f. Nervenheilk.*, 1900. Sarcoma in right island and corpus striatum.24. Broca, *Arch. gen. de Mèd.*, 1896. Tumor of chiasma, could not be found, ventricles drained, death in 2 weeks.—25. McBurney, *Med. Record*, 1896. Sarcoma of left centrum ovale and base. Death.—26. McCosh, *Med. Record*, 1896. Tumor of corpora quadrigemina. No change after operation.—27. Bury, *British Med. Journ.*, Nov., 1896. Tubercle of optic thalamus. Death in 2 months.—28. Glaser, *Langenbeck's Arch.*, i, 1901. Glioma, region of calamus scriptorius, death in ¾ hours.—29. Rohner, *Rev. Mèd. de l'Est.*, xxx, 1898. Basal tumor.—30. D'Alloco, *La rif. med.*, 1897. Optic thalamus.—31. Gordinier, *Alb. Med. Annals*, 1898. Centrum ovale, died.—32. Thomas and Hamilton, *Journ. of Exp. Med.*, 1897. Corpus callosum, died.—33. Taylor, *Journ. of Exp. Med.*, ii, 6. Optic thalamus, died.—34. Thompson and Ferguson, *Glasgow Med. Journ.*, 56, p. 424-429. Tubercle, suspected in motor area, found on autopsy in lenticular and caudate nuclei.—35. Starr, *British Med. Journ.*, Oct. 16, 1897. Large sarcoma in basal ganglia, under area exposed. Death, 2 weeks.—36. Schulze, *Deutsche Zeitschrift f. Nervenheilk.*, 1896. Gliosarcoma at right side of base with hydrocephalus of left lateral ventricle.

*Cerebellum.*—37. Ziegenweide, *Psych. en neurol. Bl.*, 1899. Suspected in cerebellum, on autopsy found between right cerebellum, tentorium and pons.—38. Heidenhain, *Arch. f. klin. Chirurgie*, 64, p. 848. Sarcoma of upper and lower worm. Death in 7 hours.—37. Stieglitz, Gerster and Lilienthal, *American Journ. of the Med. Sciences*, Vol. iii, p. 509. Gliosarcoma of right cerebellum, meningitis.—40. Borelius, *Neur. Centralbl.*, 1897. Fibroma of right cerebellum. Death soon after.—41-42. Janz, *Schmidt's Jahrb.*, 1897. Two cases, tumor cerebelli, not found, death.—43. Gerster, *American Journ. of the Med. Sciences*, May, 1896. Cerebellar tumor, not found.—44. D'Alloco, *Rif. med.*, 1897.—45. Starr, *British Med. Journ.*, Oct. 16, 1897. Glioma, right cerebellum, ventricle drained, improvement, death in 8 days.—46. Starr, *ibid.*, Cerebellar tumor, not found. Operation had to be stopped owing to hemorrhage. Discharged in same condition.—47. Descos, *Lyon Mèd.*, xcii. Not found, death.

*Region Not Stated or Indefinite.*—48. Koster and Schiller, *Neurol. Centralblatt*, 1897. Sarcoma, left hemisphere. Death, 2 months after slight improvement.—49. Gould, *Clin. Soc. of London*, Jan. 11, 1898.—50. Sidney, *British Med. Journ.*, 1898, p. 215.—51. Swolfs, *Journal de Neurologie*, 1897, No. 24.—52. Hirsche, *Wiener klin. Wochenschrift*, 1896, No. 5.—53. Nicoll, *Glasgow Med. and Chirurg. Soc.*, March 4, 1898.—54. Fisher, *Transactions of the American Neurological Association*, May, 1897.—55. Langdon, *ibid.*—56. Spencer, *Therap. Gazette*, 1898. Operated in occipital region, recovered.—57. Phillips, *British Med. Journ.*, 1898. Recovered.—58. Spencer and Keene, *Therap. Gazette*, Recovered.—59. Sinkler, *Journ. of Nervous and Mental Disease*, xxi, p. 796. Death in 54 days.—60. Hitzig, *Therap. Wochenschrift*, 1896, Sarcoma, left hemisphere, death in 3 weeks.—61. Hitzig, *ibid.* Patient's condition not changed.—62. Ruheman and Hahn, *Deutsche med. Wochenschrift*, 1896. Operated on cerebellum, position (?) No autopsy.—63. Gibson, *Edinb. Med. Journ.*, Feb., 1896.

### TUMORS FOUND, NOT REMOVED.

*Central Region.*—64. Krauss, *New York Med. Journ.*, July 30, 1898. Trephined over left arm center and found hard mass 1 inch below cortex which could not be removed. Recovery.—65. Wiener, *New York Med. Journ.*, 1898. Glioma of arm, face and speech centers, too large for removal.—66. Elliot, *Boston Med. and Surg. Journ.*, 1896. Multiple sarcomata of left motor zone, removal intended for a second operation, but death before this after slight improvement.—67. McBurney, *Med. Record*, 1896. Glioma of central convolutions which could not be extirpated. Result (?).—68. Henschen, *Mitth. aus dem Grenzgebiet der Med. und Chir.*, 1898. Too large and diffuse to be removed.—69. Henschen, *ibid.*, too large and diffuse to be removed; death in 8 months.—70. Starr, *British Med. Journ.*, Oct. 16, 1897, Glioma, motor area, could not be removed owing to hemorrhage. Death 24 hrs.—71. Eskridge, *Denver Med. Times*, June, 1896. Glioma, Rolandic area.—72. Fisher, *New York Med. Journ.*, April 16, 1898.—73. King, *Chicago Clinical Review*, April, 1897. Glioma.

*Frontal Region.*—74. Schulze, *Deutsche Zeitschrift für Nervenheilkunde*, 1896. Gliosarcoma of right frontal lobe. Operation interrupted owing to hemorrhage. Death next day.—75. Patel, *Lyon Méd.*, xciii, 830. Sarcoma.

*Parietal Region.*—76. Nonne, *Neurolog. Centralblatt*, 1899, p. 229. Subcortical sarcoma, left paracentr. lobe. Death 5 months.

*Cerebellum.*—77. Scheede, *Deutsche med. Wochenschrift*, 1900, No. 30, Cystic glioma in worm. Death.—78. Saenger, *Neurolog. Centralblatt*, 1899, p. 1117. Tumor between pons, medulla and cerebellum, slight improvement.—79. Saenger, *ibid.*, Partial recovery.—80. Joffé, *Deutsche med. Wochenschrift*, 1897, No. 5. Fibrosarcoma of right cerebellum. Operation interrupted on account of collapse. Death in 12 hours.

*Region Indefinite or Not Stated.*—81. Jolly, *Berl. klin. Wochenschrift*, 1899, No. 29, Glioma, too diffuse for removal. Death, 2½ years.—82. Colman and Ballance, *British Med. Journal*, March 11, 1896, Cystic glioma in centrum semiovale. One cyst evacuated. Death in 10 months.—83. Labbè, *Bull. et Mem. de la Soc. Anat.*, 55x, 9, 702, Glioma.

### TUMOR REMOVED. RECOVERY.

*Central Region.*—84. Leszynsky, *Med. Record*, Sept. 28, 1901. Endothelioma of right arm and leg center.—85. Hoppe, *Journ. of the Amer. Med. Assoc.*, Feb. 21, 1901. No recurrence after 7 years. No complete cure. Right foot and arm center.—86. Hoppe, *ibid.* Blood cyst from dura over left leg center. Died 18 months in epileptic attack.—87. Mayde, *Wiener klin. Rundschau.* Small cyst, finger center.—88. Heidenhain, *Archiv für klinische Chirurgie*, 64, p. 848. Subcort. cystic sarcoma in right arm center.—89. Treyer, *Rev. méd. de la Suisse romande*, 1900, 5 and 6. Tubercle, right face and arm area. Slight paresis remaining.—90. Treyer, *ibid.* Tubercle, slight paresis remaining.—91. Krauss, *New York Med. Journ.*, July 30, 1898. Sarcoma, left leg center. Death, 2 weeks.—92. Krauss, *ibid.* Infiltrating glioma at junction of leg and arm center, only partly removed. Death, 3 weeks,—93. Oliver and Williamson, *British Med. Journ.*, Nov. 26, 1898. Sarcoma fissure of Rolando. Recurrence.—94. Oliver and Williamson, *ibid.* Subcortical angioma in lower part of left motor region.—95. Stieglitz, Gerster and Lilienthal, *Centralbl. f. Chirurg.*, 1897. Sarcoma, left motor area, recovery.—96. Duncan and Maylord, *Glasgow Med. Journ.*, 1897. Sarcoma of right motor area, improvement.—97. McBurney, *Med. Record*, 1896. Cysts in arm center. Improvement, then death in 6 weeks due to recurrence.—98. Seydel, *Centralbl. f. Chirurg.*, 1896. Fibroma in dura opposite left arm center. Recovery with paresis in leg.—99. Hirsche, *Centralbl. f. Neurol.*, 1896. Diffuse sarcoma of left motor zone. Partial excision, slight improvement.—100. Devic and Courmont, *Rev. de Méd.*, 1897. Glioma.—101. Sick, *Deutsche med. Wochenschrift*, 1897, No. 2. Tubercle at lower end of gyrus postrolandicus.—102. Cabot, *Bost. Med. and Surg. Journ.*, June, 1897. Cyst punctured twice in 1¼ years.—103. Championniere, *Wiener klin. Rundschau*, 1897, No. 49. Sarcoma of central convolutions, improved.—104. Krönlein, *Beiträge z. klin. Chir.*, 1896, p. 253. Tubercle in left arm center.—105. Czerny, *Deutsche med. Wochenschrift*, 1896, No. 11. Cystic sarcoma. motor area.—106. Morison, *British Med. Journ.* Subdural cyst, motor area.—107. Voisin, *XII. Internat. Med. Congr.*, Aug. 24, 1897. Tumor of central convolutions.—108. Washburn and Lane, *Clin. Soc. of London*, March 17, 1897. Endothelioma, left motor region.—109. Friedlander and Schlesinger, *K. k. Gesellsch. f. Aerste in Wien*, Jan. 21, 1898. Gumma, lower end of anterior central lobe.—110. Doyen, *27. Congress der deutsch. Gesell. f. Chir.* Tumor of central convolution.—111. Carriere, *Congrès de Neurolog. Toulouse*, Aug., 1897. Neuroglioma of motor area.—112. Eliot, *Boston Med. and Surg. Journ.* Cyst in lower arm center, evacuated. Death in 3 months.—113. Bonhoeffer, *Monatschr. f. Psych.*, 1898, No. 3. Glioma, upper one-third of right central. Death, 3 months.—114. Dallas and Mongeri, *Gaz. med. d'Orient*, 1897. Sarcoma, lower end of the left postcentral convolution. Death in 4 months of cachexia.—115. Zenner, *Transactions of the American Neurological Association*, May, 1897. Tumor of central convolution. Death 9 months after second operation.—116. Abrahams and Tait, *Occid. Med. Times*, June, 1896. Recovery.—117. Armstrong, *Montreal Med. Journ.*, May, 1896. Cyst, recovered.—118. Braymer, *Charlotte Med. Journ.*, Nov., 1896.—119. Corine and Doyen, *Bull. de la Soc. d. l. Anat. de Paris*, Dec., 1897. Improved.—120. Crowley, *Transact. Medical Soc. of Calif.*, 1898. Sarcoma, improved.—121. Dreyfus, *Lyon Méd.*, 1898. Gumma, improved.—122. Eskridge and Parkhill, *Méd. News*, 1896. Cyst, improved.—123. Eskridge and Parkhill, *ibid.* Angioma, improved.—124. Grisson, *Neurol. Centralblatt*, 1898. Cyst, recovered.—125. Hossler, *Neurol. Centralblatt*, 1897. Improved.—126. Mareau, *Arch. med. d'Angres*, 1897. Improved.—127. Mayer, *Annals of Surgery*, 1898. Cyst, improved.—128. Nedwill, *Lancet*, 1898. Hydatid, improved.—129. Stratton and Crowley, *Pac. Med. Journ.*, 1898. Sarcoma, improved.—130. Tauber, *Arch. de Psych. russ*, 1896. Improved.—131. Putnam and Richardson, *Boston Med. and Surg. Journ.*, cxl, p. 129. Sarcoma, motor area, not found at first but removed at second operation at same site. Death, 6 months.—132. Gibson, *Trans. of Med.-chirurg. Soc. of Edinburgh*, N. S., xv, p. 27. Glioma, motor area.—133. Sinkler, *Journ. of Nervous and Mental Disease*, xxi, p. 796. Trephined at right facial center, not found, but found and partly removed at second operation. Death, 1 month.—134. Morestin, *Bull. et Mem. de la Soc. Anat.*, 65 i, 249. Sarcoma of cranium, pressing on motor area.

*Frontal Region.*—135. v. Wagenburg, *Psych. en neurol. Blad.*, 1898. Sarcoma, recovery with slight paralysis.—136. Esteves, *Prog. Med.*, Dec. 23, 1899. Hydatid cyst, recovery.—137. Graff, *Deutsche med. Wochenschrift*, 35, 1899. Cyst, recovery after evacuation.—138. Thomas and Keene, *Amer. Journ. of the Med. Sciences*, Nov., 1896. Gliosarc, recovery with facial paresis and amaurosis.—139. Rossolimo, *Arch. f. Psych.*, xxix, p. 528. Cystic gliosarcoma, death 10 months from recurrence. — 140. Naumann, *Hygiea*, 58, p. 155. Sarcoma periost. cranial, pressing on frontal lobe.—141. Carle, *Rev. de Chirurg.*, Feb., 1899. Sarcoma, recovery.—142. Tubenthal, *Deutsche med. Wochenschrift*, xxv. Cyst, evacuation, recovery.—143a. Fescher, *Deutsche med. Wochenschrift*, xxiv, 52. Gumma, recovery.—143b. v. Bergman, *Berliner klin. Wochenschrift*, 38, p. 219. Tumor, frontal region; recovery.

*Parietal Region.*—144. Clarke and Lansdowne, *British Med. Journ.*, April 19, 1901. Two tumors (encaps. sarcoma) removed within a few weeks.—145. Heidenhain, *Arch. f. klin. Chir.*, vol. lxiv, p. 848. Tubercle, paracentr. lobe, recovery.—146. Bayerthal, *Münch. med. Wochenschrift*, 1899. Tubercle, paracentral lobe, much improvement.—147. Richardson, *Boston Med. and Surg. Journ.*, 1896. Removed on second trephining.—148. Brissaud, *Bullet. de Chir.*, May, 1896. Tubercle posterior end of the left Sylvian fissure.—149. Starr, *British Med. Journ.*, Oct. 16, 1897. Fibrocyst, inferior parietal lobe. Recurrence in 4 months.—150. Mills and Keene, *Journ. of Nervous and Mental Disease, 27*, p. 244. Endothelioma, superior parietal convolution.—151. Pitt and Lane, *Clin. Soc. Transact.*, 29, p. 164. Sarcoma, recovery.—152. King, *Chic. Clin. Rev.*, April, 1897. Sarcoma, left superior parietal region, recovery.—153. Walton, *Boston Med. and Surg. Journ.*, 144, p. 205. Subcortical parietal cyst, recovery.

*Temporal Region.*—154. Hoppe, *Journ. of Amer. Med. Assoc.*, Feb. 21, 1901. Cyst, first temp.-sphenoid lobe. Recovery with slight aphasia.—155. McCosh, *Med. Record*, 1896. Angioma of pia in left temporal region. Complete recovery.—156. Broca, *Centralbl. f. Chir.*, 1897, Neuroglioma of left temporal lobe. Cure.—157. Hobart, *K. k. Gesell. f. Aerzte in Wien*, June 17, 1898. Gumma, right temporal region.—158. Barr and Nicoll, *British Med. Journ.*, Oct., 1897. Sarcoma in temporal region. Death in 2½ months.—159. Nicoll, *Lancet*, Oct. 29, 1898. Sarcoma originating from middle ear; intracranial portion removed. Death in 2½ months.

*Occipital Lobe.*—160. Hoppe, *Journ. of Amer. Med. Assoc.*, Feb. 2, 1901. Glioma, left occipital lobe. Well after 3 years.—161. Kocher, *Schweiz. Correspbl.*, 27, p. 397. Osteoma growing from occipital bone; recovery.

*Deeper Parts of Brain.*—Heidenhain, *Archiv f. klin. Chir.*, vol. lxiv, p. 848. Melanosarcoma of tela choroid and ependyma of descending cornua. Entire right temporal lobe extirpated.—163. Bastianelli, *Boll. de la Soc. Lancis*, p. xv, 1. Sarcoma of anterior fossa pressing eyeball forward.

*Cerebellum.*—164. Schede, *Deutsche med. Wochenschrift*, 1900, No. 30. Glioma, recovery.—165. Collins and Brewer, *Med. Record*, May 15, 1897. Subcortical right cerebellar tubercle, partly removed. Death, 2½ months.—166. Guthrie, *Practitioner*, 1898. Cerebellar cyst, improved.—167. Moran and Kerr. *Virg. Med. Semi-monthly.* Cyst, improved.—168. Gibson, *Transac. Med. Chirurg. Soc., Edinburgh*, N. S., xv, 27. Fibrosarcoma cerebellum, removed; recovery.—169. Murri, *Lancet*, 1897. Fibrosarcoma, left cerebellum. Incomplete removal, slight improvement.—170. Parkin, *British Med. Journ.*, 1896, p. 1776. Glioma; recovery.—171. Bevor, Ballance and Lumm, *Brain*, 1897. Fibrosarcoma, anterior part, right cerebellum.—172. v. Bergmann, *Berliner klin. Wochenschrift*, 38, p. 219. Cerebellar cyst; recovery.

*Region Indefinite or Not Stated.*—173. Ziehe and Roth, *Deutsche med. Wochenschrift*, 1897, 19. Perithelioma.—174. Weissgerber, *Münch. med. Wochenschrift*, 1896, No. 16. Cyst in cortex.—175. Kosynski, *Medyzyna*, 1898, No. 1-3, 5.—176. Lundmark, *Ups. Lok. For.*, 1898. Cancer, not improved.—177 O'Hara, *Australas. Med. Gazette*, 1898. Hydatid, recovered.—178. Vance, *Australas. Med. Gazette*, 1898. Hydatid, improved.

### VI. TUMOR REMOVED, DEATH.

*Central Region.*—179. Hirschfelder, *Pac. Med. and Surg. Journ.*, April, 1896. Glioma of right post-central gyrus. Death, 7 days.—180. Breganski and Wrzesmowski, *Centralbl. f. Chir.*, 97. Sarcoma of left Rolandic area. Died of meningitis.—181. Schulze, *Deutsche Zeitschrift f. Nervenheilkunde*, 1896. Sarcoma of left anterior central convolution.

Death of hemorrhage.—182. Stieglitz, Gerster and Lilienthal, *Centralbl. f. Chir.,* 1897. Sarcoma in left arm, face and speech center. Death from hemorrhage.—183. Ballance, *Med.-Chir. Transact.,* vol. lxxix. Cystic angiosarcoma, left arm center. Death due to hemorrhage. Had been evacuated four times and removed once before.—184. Dinkler, *Wanderversamml. der Südwest-deutschen Irrenärzte,* May 22, 1899. Fibroma, death in 12 hours.—185. Booth, *Transactions of the American Neurological Association,* May 6, 1897. Tumor, left leg center. Death in 4 hours from hemorrhage.—186. Ziehen, *Zeitschr. f. prakt. Aerzte,* 1898, No. 1. Fibroma, left motor zone. Death, 8 days from meningitis.—187. Hawkes, *Intercolon. Med. Journ.,* Dec., 1897. Hydatid cyst, left fissure of Rolando. Death in 3 days.—188. Schnitzler, *Centralbl., f. d. g. Therap.,* Feb., 1898. Tubercle, right arm center. Death in 26 hours of edema of brain.—189. Taylor and Eliot, *Boston Med. and Surg. Journ.,* 1896. Endothelioma.—190. Starr, *British Med. Journ.,* Oct. 16, 1897. Sarcoma of left upper motor area, death 6 hours, hemorrhage.

*Frontal Region.*—191. Taylor, *Boston Med. and Surg. Journ.,* Jan., 1896, Endothelioma left frontal region. Death in several hours,—192. Starr, *British Med. Journ.,* Oct. 16, 1897. Cyst, right upper frontal region, removed, recurred; infection with death after second operation.—193. Starr, *British Med. Journ.,* Oct. 16, 1897. Cyst, frontal region, evacuated. Death in 6 days.

*Parietal Region.*—194. Rychlensky, *Medyzyna,* 1897, 1 and 2. Glioma in paracentral lobe. Death two days after evacuation of cyst.—195. Steffenson, *Med. News,* lxxvi, p. 175. Tumor in angular, supramarginal and first temporo-sphenoidal lobe. Death in 2 hours.

*Temporal Region.*—196. Stewart, *Northwest Lancet,* 1897. Sarcoma.

*Deeper Parts of Brain.*—197. Dinkler, *Wanderversamml. der Südwest-deutschen Irrenärzte,* May 22, 1899. Tumor in white substance of left cerebrum. Death of shock.—198. Krogius, *Rev. de Chirurg.,* 16, p. 434. Endothelioma of middle fossa; death.

*Cerebellum.*—199. Pershing, *Med. News,* 1898. Glioma.

*Region Indefinite or Not Stated.*—200. Hitzig, *Therap. Wochenschrift.* Large endothelioma of dura. Death in collapse.—201 and 202. Lundmark, *Ups. Lok. For.,* 1898. Two cases; died soon after operation.

Submitted for publication, Jan., 1903.

# A CONTRIBUTION TO THE PATHOLOGY OF PARAMYOCLONUS MULTIPLEX (FRIEDREICH'S TYPE).*

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The peculiar type of muscular contractions described by Friedreich under the name of paramyoclonus multiplex is of very rare occurrence. The uncertain mechanism of its production and the controversy attending its classification and position among the motor neuroses warrant a brief review of the literature as an introduction to the clinical and pathological report which is to follow.

*Historical.* The history of paramyoclonus multiplex began in the year 1881 with the description of a case by Friedreich,[1] presenting the following peculiarities: The patient was a man with lung tuberculosis, aged fifty, who, following a violent fright, developed quick clonic contractions of the muscles of the proximal segments of the upper and lower extremities. The contractions resembled in character those induced by the electric current, the individual muscles springing forward as though excited by an invisible electrode. It was particularly noted and emphasized that individual muscles, such as the sartorious and supinator longus, would spring forward in independent contractions which singly are incapable of voluntary innervation. Moreover, there was an absence of synergetic muscular action, so that the locomotor effect attending such coördinated muscular contraction was absent. When the contractions were very strong, however, a slight movement resulted. Occasionally, owing to the great rapidity of the contractions, the muscle was thrown into a state of momentary tetanus. There was cessation of the movements during sleep. These muscle contractions were increased by nearly all forms of peripheral and mechanical stimuli. They were diminished or entirely inhibited by voluntary movement, so that little inconvenience was caused by performing acts of everyday life, in this respect presenting a marked contrast to the coordinated movements of cerebral origin. The knee-jerks were greatly exaggerated. The gross motor power suffered no impairment. The electrical reactions were normal. This was essentially true of the psychical and sensory functions. At times painful flexor spasms of the thigh occurred. There were absolutely no stigmata of hysteria.

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The peculiar features of this affection, viz., the spontaneous contraction of individual muscles not singly under the control of the will, and the absence of synergetic muscular action in the affected members, were so at variance with the known characteristics of muscular contractions of cerebral origin as to render some other explanation probable. Friedreich, after a careful consideration of all the possible sources, as the brain, spinal cord, nerves and muscles, proposed the hypothesis which referred the symptoms to an *excessive irritability of the anterior horn cells of the cord.* This theory was in harmony with the special clinical features of the case, i.e., the single muscle contraction, the absence of synergetic action, the great exaggeration of the knee-jerks, and their diminution as the motor symptoms subsided, the great susceptibility of the contractions to peripheral stimuli, and their cessation on voluntary innervation of the part.

In the course of a few years the accuracy of Friedreich's observation was confirmed on many sides by notable observers. Among the earlier recorded cases were those of Löwenfeld,[2] Remak,[3] Seeligmüller,[4] Francotte,[5] Homên,[6] Allen Starr,[7] Bechterew[8] and Marie.[9] It was but natural that Friedreich's description, based on one case, should undergo considerable enlargement and some variation, as the literature was enriched by other observers, and that even new forms and types should be added. Thus, in 1886, Seeligmüller described a congenital case; Unverricht[10] in 1891 added a family form associated with epilepsy, and Gucci[11] an hereditary form in 1893. Some of the more important semeiological deviations from the original Friedreich type which have been subsequently described are as follows: That the muscular contractions are *not* limited to the proximal segments of the extremities, but that all the voluntary muscles may be involved with the sole exception of those moving the eyeballs.

Voluntary movement has not in all cases seemed to soothe or interrupt the contractions; indeed, the contrary effect has been noted. In some cases peripheral stimuli have failed to increase the symptoms. The knee-jerks, although usually exaggerated, have been found occasionally of normal intensity or even diminished. In two cases they were absent. The electrical reactions have been essentially normal. In a few cases only was a slight qualitative increase noted. Rarely contractions have persisted during sleep. Pains and paresthesias have been observed accompanying and following the paroxysms. Fright as an etiological factor was by no means constant, and a variety of predisposing and exciting causes are on record.

*Classification.* But a small proportion of the cases now reposing in the literature under the title paramyoclonus multiplex or its abbreviated form, myoclonus multiplex, are typical examples of this affection. Many cases are hardly recognizable if Friedreich's conception is adhered to, some observations having more in common with convulsive tic, maladie de tic, chorea and hysteria. In some observations the peculiar features which led Friedreich to separate this from the other motor neuroses, i.e., the isolated contraction of a muscle not singly under voluntary control and the absence of locomotor effect, have been completely ignored. Cases are recorded as paramyoclonus multiplex in which the affection was characterized by convulsive movements of a paroxysmal character very far indeed from the individual shock-like muscle contraction which usually first becomes apparent when the surface of the body is exposed, and which interferes but little with voluntary movement. This statement will receive more weight when the tabulations of Unverricht are considered, who collected and classified the cases recorded up to the year 1891. Of the forty-eight cases collected, Unverricht accorded to only six the dignity of paramyoclonus multiplex, and to eleven cases a resemblance only to the paramyoclonus; eleven cases he considered uncertain, and the remainder were grouped with hysteria, chorea, or the tic forms.

A survey of the more recent literature shows the same confusion to prevail. Nearly all forms of myoclonia in the broadest sense of the term are included under this head, while the pure cases of paramyoclonus form but a small minority.

It must be observed, however, that muscular contractions identical with or closely resembling those of the essential form of paramyoclonus are found associated with or dependent upon other conditions. Thus, in hysteria and the traumatic neuroses, twitchings resembling paramyoclonus are recorded; and as a sequela of joint affections and then presumably of reflex origin. They are also described in conjunction with epilepsy, the so-called myoclonus epilepsy. Rarely muscular spasms of an entirely different type, i.e., the tic convulsif, maladie de tic, are accompanied by movements of a paramyoclonic nature.

In the other forms of myoclonia the Friedreich type may be very closely simulated, but attention to the characteristics just outlined usually renders differentiation possible. Dejerine[12] emphasizes the strong resemblance which some hysterical movements bear to this affection. Indeed, some observers, as Moebius,[13] regard paramyoclonus as a manifestation of hysteria. Moebius, in support of the hysterical nature of the affection, mentions the difficulty incurred in determining the contraction of an isolated muscle not individually under voluntary control when such contractions are lightning-like. This may be true of the periods of great exacerbation, but would hardly be applicable to the whole course of the disease. Furthermore, it is impossible to reproduce these contractions at will, which, according to our present conceptions, would exclude hysteria. Schultze[14] regards the paramyoclonus multiplex as a generalized form of tic convulsif. Many observers, as Oppenheim,[15] Unverricht, Risien-Russell,[16] Allen Starr and Wollenburg,[17] view this affection as an independent form of motor neurosis. Others, as Raymond,[18] would fuse the various myospasms together under a generic term as myoclonia, considering them as allied in nature and differing only in degree, the degenerative tendency representing the common basis and essential factor in the production of them all. This would include the maladie de tic, tic convulsif, electric chorea of Henoch-Bergeron, paramyoclonus multiplex, myokymia, and fibrillary twitchings.

*Pathogenesis.* Diversity of opinion is not confined to the classification of this affection, but the origin and the mechanism of its production are the subject of controversy. Pathological anatomy has thrown no light upon the subject. The theories which today receive serious attention are those of the cerebral and the spinal origin. Whether the latter consists of a derangement of the motor portion of the reflex arc (Friedreich) or of the sensory portion, as advocated by Vanlair,[19] is secondary to the question at issue.

According to our present knowledge, the evidence is strongly in favor of a spinal origin of the Friedreich type of paramyoclonus multiplex. The electrical stimulation of the motor cortex with the most delicate electrode fails to produce a contraction of an individual muscle. On the contrary, the result is a movement, a synergetic muscular action. We may assume with a certain degree of probability that movements rather than individual muscles are here represented. The medullary centers, on the other hand, have a more individual relation with the respective muscles under their control, although the exact nature of this relation is still a subject under investigation. It will be remembered in this connection, however, that Sano[20] advocates an individual muscle representation in the anterior horn cells, based on experimental studies.

Those rare cases in which paramyoclonic twitchings have occurred in the course of organic disease of the cerebral cortex must be considered in this relation. Examples of such are paresis, chronic meningitis with cortical atrophy (Murri[21]), chronic uremic edema of the cortex (Levi and Follet[22]), and myoclonus epilepsy (Unverricht, Clark and Prout[23]). These cases are by some observers considered confirmative of a cortical origin. If, however, minute pathological changes of the cerebral cortex were capable of inducing this type of muscular contraction, they should be of more frequent occurrence, considering the vast number of such conditions, constantly under observation. In view of this rarity, a coëxisting alteration of the spinal centers is more probable; and in the family affection, characterized by the association of myoclonus and epilepsy, we are dealing with a degenerative affection of the cerebrospinal axis, the epilepsy referable to the upper, the myoclonus to the lower centers. The few cases of hemimyoclonus (Minkowsky,[24] Bernhardt,[25] and Seeligmüller[26]), usually cited as evidence of the cerebral origin, weigh but lightly in the balance.

Very interesting and suggestive from an etiological point of view and corroborative of the spinal theory are those cases of paramyoclonus multiplex occurring in conjunction with articular affections. Chauffard's[27] case of hemiparamyoclonus followed directly on an attempt to break up old joint adhesions in the right knee and hip. Levi and Follet report a case of paramyoclonus multiplex complicating spondylose rhizomelique with the usual joint manifestations. The articular muscular atrophies and the old theory offered in explanation, the perversion of the functions of the anterior horn cells (Paget, Charcot) arise in this connection. Raymond[28] demonstrated experimentally that if the posterior roots are cut before the articulation is disturbed, this atrophy will not occur. This is almost indisputable proof of its reflex origin. If pathological reflex stimuli from the articulation can affect the trophic function of the anterior cells, is it not reasonable to infer that the motor function may be perverted in a similar manner with a resulting hyperexcitability? It is a common observation that the tendon jerks are exaggerated in muscles which are the seat of this atrophy, the knee-jerks in affections of the knee joint, the Achilles jerk in ankle joint disease. This exaggeration would hardly be expected in atrophic muscle groups unless accompanied by an unusual irritability of the reflex centers. Gowers[29] cites an interesting case in this connection. A young man, following an injury to the left knee, developed an acute atrophy of the quadriceps extensor. When seen two years later the atrophy still persisted, the left knee-jerk was exaggerated, and there was in addition a well-marked ankle clonus upon the left side. This same condition was persistent one and a half years later, and with the associated ankle clonus would seem to prove that the functional and trophic changes in the anterior horn cells in joint affections may not only be of long duration, but may extend to neighboring centers. The muscles from a case of articular atrophy were examined by Darkschewitz[30]. A majority of the muscle fibers were found to have undergone a diminution in size, averaging about half the normal diameter. These atrophic changes in the muscle fibers, in an affection where presumably the trophic function of the anterior horn cells has been restricted by some inhibitory influence, receive additional significance from the muscle changes found in my case of paramyoclonus multiplex, an affection presumably due to an exaggeration of the function of the anterior horn cells. Here the diameter of the muscle fibers was two or three times in excess of the normal.

As further evidence in favor of the spinal origin of the peculiar contractions under consideration may be mentioned the relationship existing between paramyoclonus multiplex, myokymia and fibrillary twitchings. Between the fascicular contractions and the muscle waves and the individual muscle contractions of paramyoclonus there are transition forms: a series of gradations, so that one condition merges imperceptibly into the other. The myoclonus fibrillaris multiplex of Kny[31] represents such a transition form. Thus, in one case the affection may bear the stamp of a myokymia, yet simple fibrillary waves occurring and occasionally the whole muscle undulating; so in paramyoclonus certain undulations may occur. Dana,[32] in a recent communication, emphasizes the relationship of myokymia and paramyoclonus multiplex. The dependence of myokymia and fibrillary contractions on disease of the lower nervous mechanism is fairly well established, and has been recently discussed by Walton.[33] The etiology in some of the recorded cases (cited by Walton): lead-intoxication (Huber); following in the wake of old poliomyelitis (Williamson, Walton), low grade neuritis (Biancione), sciatica (Gowers), bear no other interpretation. This receives additional weight from the accompanying pains, paresthesias and electrical disturbances not infrequently observed.

There is still another peculiarity observed in these three forms of muscle contraction, and that is the passive attitude manifested by the patient towards them. The muscle contractions and undulations are felt as such, but otherwise cause little or no disturbance, save occasionally a cramp or a pain. This is largely accounted for by the usually complete preservation of the power of performing voluntary acts and the absence of strong convulsive movements of a coördinated type. It is also possible that their origin in the lower centers, far removed from the psychical sphere, may also play a rôle.

*Remarks.* I. The term paramyoclonus multiplex, or myoclonus multiplex, should be reserved for that form of myospasm characterized by multiple, spontaneous, isolated contractions of individual muscles.

II. This type is peculiar and distinctive, and receives its most logical explanation in a disturbance of the spinal centers.

III. This type should be carefully separated from the cerebral type of the myospasms which are characterized by movements of a more or less coördinated type, as are observed in the maladie de tic, tic convulsif and the convulsive tremor of Pritchard and Hammond.

IV. The contractions of paramyoclonus multiplex are closely related to the myokymia and fibrillary contractions.

V. Paramyoclonus multiplex may occur as an idiopathic or a deuteropathic affection, in the latter complicating various organic and functional diseases of cerebral and spinal origin.

## CASE REPORT.*

These observations were made in the surgical wards of Bellevue Hospital, service of Dr. B. B. Galludet, through whose courtesy I was permitted to study and publish the case.

*History.* July, 1901. The patient was a man, aged forty-nine years, Hungarian by birth, tailor by occupation. His admission to the surgical ward was for the treatment of a tuberculous affection of the left ankle joint, which was discharging from several sinuses. An operation had been performed upon the joint at the New York Hospital a few months before. This was his sole complaint on admission, and the surgeons discovered by accident the curious muscular contractions with which he was afflicted and which apparently caused him no concern. It was ascertained that these had made their appearance about six months before without pain or paresthesia, first in the upper, soon after in the lower extremities. There had been no preceding illness except the joint affection before alluded to. He was of moderate habits, denied venereal disease, and no family neurotic taint was discoverable, but owing to his long exile from Hungary his knowledge in this direction was imperfect. No member of his family, to his knowledge, had ever had a convulsion. A few months before the onset of the disease, being poor and in feeble health, he had written to Hungary requesting money from his family with which to return home. This had been promised him, but in due time instead of a money order he received a letter containing a refusal. He despaired of ever being able to return to his native land, became sad and depressed, and a few weeks later the twitchings began. As he related this story his eyes filled with tears, and on subsequent allusion to it he always showed profound emotion. The twitchings had not interfered with the ordinary acts of life, as dressing, walking or holding a newspaper, and had caused him but little annoyance; toward the end of the day he would feel rather fatigued.

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*Read, with presentation of specimens, before the New York Neurological Society, February, 1903.

*Physical Examination.* The man is of medium height, well made, body weight well preserved. Facial expression is quiet and melancholy. Skin pallid, mucous membrane a fair color. The face free from tremor or movement of any kind. Dressed and sitting in a chair, as when I first saw him, no abnormal movements of any kind were visible, except an occasional playing of the fingers. Locomotion and station were naturally somewhat interfered with by virtue of the joint affection, but were entirely unaffected by the muscular contractions. The pupils and pupillary reactions were normal, as were all the cranial nerves. No nystagmus. After removal of the clothing, the picture presented by the patient was a strange and unusual one. The muscles of the arms and shoulder girdles, thighs and buttocks displayed the greatest activity, individual muscles springing forward in contraction with the greatest rapidity and variation; sharp, shock-like contractions following one another with regularity and rhythm. The whole body of a muscle contracted as if stimulated by an invisible electrode. These contractions were unaccompanied by any locomotor effect. Occasionally, a barely perceptible flexion, extension or supination resulted from a particularly violent or prolonged contraction. It was further noted that while symmetrical muscles were involved, the contractions were by no means synchronous or isochronous. The movements were all clonic, no tonic contractions being noted. Contractions were noted in the following muscles: Deltoids, pectorals, scapular group, biceps, triceps, supinators, gluteal groups, extensors and flexors of the knee, and the sartorius; an occasional contraction was noted in the forearms and in the calves of the legs. The greatest play of movement was in the proximal segment of the extremities. The abdominal muscles and the diaphragm were quite free. The intensity and rapidity of these contractions could be modified in various ways. Mechanical stimulation of the skin and muscle increased them, while a coördinated act, such as grasping or picking up an object, caused their cessation or a very marked diminution. In walking and standing this diminution in the lower extremities was very noticeable. The contractions were most severe in the recumbent posture, less while standing, and almost disappearing while walking to and fro. An unusual and important peculiarity of these spasms was the contraction of individual muscles, as the sartorius and supinator longus, the isolated contraction of which cannot be produced at will. There were apparently isolated contractions of the brachialis anticus and coraco-brachialis, but this could not be determined with certainty. The muscular system was quite well developed and showed no evidence of pathological hypertrophy or atrophy. The gross motor function was undisturbed. The myotatic irritability was increased. No electrical examination was made. Sensation was undisturbed. The tendon and skin reflexes were all present and very active; the knee-jerks especially were much exaggerated. Flexor response to plantar stimulation. No stigmata of hysteria.

The man was quiet and uncomplaining and shunned the society of his fellow-patients. He presented no evidence of mental enfeeblement. A few weeks later acute symptoms developed, death ensuing apparently from a generalization of the tuberculous process; as the thoracic and abdominal cavities were not opened, the immediate cause of death must remain unknown. It is interesting to note that the muscular contractions persisted the whole time during which he was under observation, varying, however, in intensity, and, according to the statement of the house-surgeon, were observed for twenty minutes after the respiratory and cardiac functions had ceased. According to the nurse's statement, the contractions ceased during sleep.

*Histological Examination.* The autopsy was performed forty-eight hours after death. The brain, spinal cord, and the right radial and anterior crural nerves were removed and placed in 10 per cent formalin solution. Portions of the right supinator longus, right sartorius, and left trapezius muscles were also removed.

*Brain.* Sections were prepared from the Rolandic area on both sides and from the frontal, temporal and occipital lobes, according to the Nissl, Weigert-Pal, Marchi and Van Gieson methods. No evidences of pathological changes were discoverable in nerve structures, meninges, vessels or glia. The outlines of the cortical cells and their processes are well preserved. A few cells are moderately pigmented. The nuclei, nucleoli and their refractive bodies are quite distinct. The nuclear network, however, is broken up and appears granular and homogeneous, remaining unstained or receiving a faint bluish tint. The Nissl granulations are coalescent and rather clumpy, staining intensely blue. The slight alterations in the nuclei and Nissl bodies are dependent on cadaveric changes.

*Spinal Cord.* Sections were prepared from various levels of the cord by the Nissl, Weigert-Pal, Marchi and Van Gieson methods. They were essentially normal. The spinal stichochromes of the anterior horns were absolutely normal. Cell bodies, processes, nuclei and nucleoli, no excess of pigmentation. The Nissl granulations, while somewhat coarse and rounded, are normal in distribution and arrangement. The vessels of the gray matter of the cord, especially in the cervical and upper dorsal regions, are distinctly thickened. The vessels in the lateral columns are also slightly sclerosed. This thickening involves chiefly the adventitia; nowhere was obliteration of the lumen observed.

The capillaries of the brain and cord are distended with blood.

*Peripheral Nerves.* The peripheral nerves, the inter-muscular nerve fibers and the muscle spindles were normal.

*Muscles.* The muscles were fixed and hardened in formalin, alcohol and Müller's fluid, and stained by the carmine, hematoxylin, Van Gieson and the Marchi methods. The supporting structures (endomysium and perimysium) were normal, as were the blood vessels.

The picture presented by the muscles on transverse section is striking, the diameter of the muscle fibers reaching an unusual size (see Figs. 1 and 2). Of the larger fibers which form the majority in any given field, the diameter averages 150 micromillimeters and, in many, 175 micromillimeters. In contrast to this are very small fibers having crescentic forms or flattened out on the periphery of the large fibers. Of these smaller fibers some are below the normal standard. The fields of Cohnheim are quite distinct. Instead of finding the nuclei of the sarcolemma sheath confined to the periphery of the muscle fiber and just beneath this membrane, as is customary in man, they are found scattered as well between the sarcous elements (Fig. 3). This is not true of all the fibers, but is observed in a large number and in all sections studied. Sometimes as many as three or four nuclei occupied a

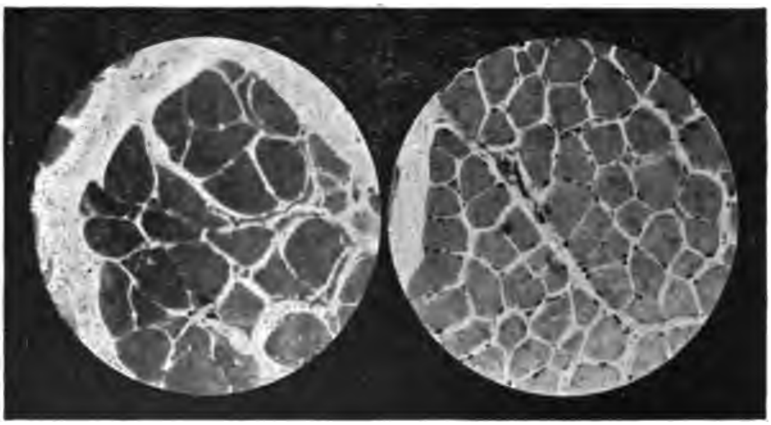

Fig. 1. Fig. 2.

Fig. 1—Right supinator longus muscle from case of paramyoclonus multiplex.

Fig. 2—Normal muscle. Same enlargement as Fig. 1.

central position in a single fiber, usually only one or two. They are identical in staining properties with those of the sarcolemma sheath. On longitudinal section the transverse striation is distinct and well preserved (Fig. 4). In the smaller fibers only is it at times indistinct or absent, these showing in addition a tendency to longitudinal cleaving. The nuclei of the sarcolemma sheath are increased in number; their structure and size are normal. Small clumps of a dark brown or yellow pigment are observed scattered over both the transverse and longitudinal sections, in the latter especially in relation with the sarcolemma nuclei. No degeneration and no vacuolization of the sarcous substance is present. No degenerations were demonstrable by the Marchi method.

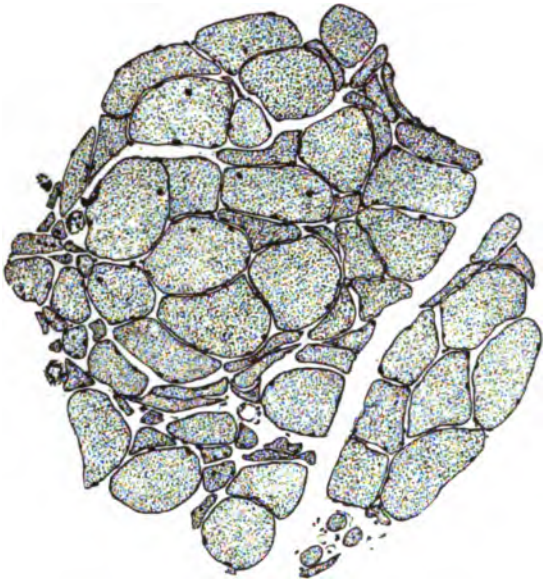

Fig. 3—Sartorius muscle. Paramyoclonus multiplex. Fibers hypertrophied with centrally situated nuclei.

*Remarks.* The peculiar character of the muscular contractions in the case just described entitles it to a place among those rare cases of essential paramyoclonus multiplex. The histological examination of the spinal cord and biceps muscle from the original Friedreich case was carried out by Schultze.[34] The result was an entirely negative one. This and my own case constitute, I believe, the only examples on record of this type of paramyoclonus multiplex with systematic histological examination. The examination of the nervous system in my case was entirely normal. The muscle fibers, on the other hand, while retaining their normal structure, were considerably hypertrophied.

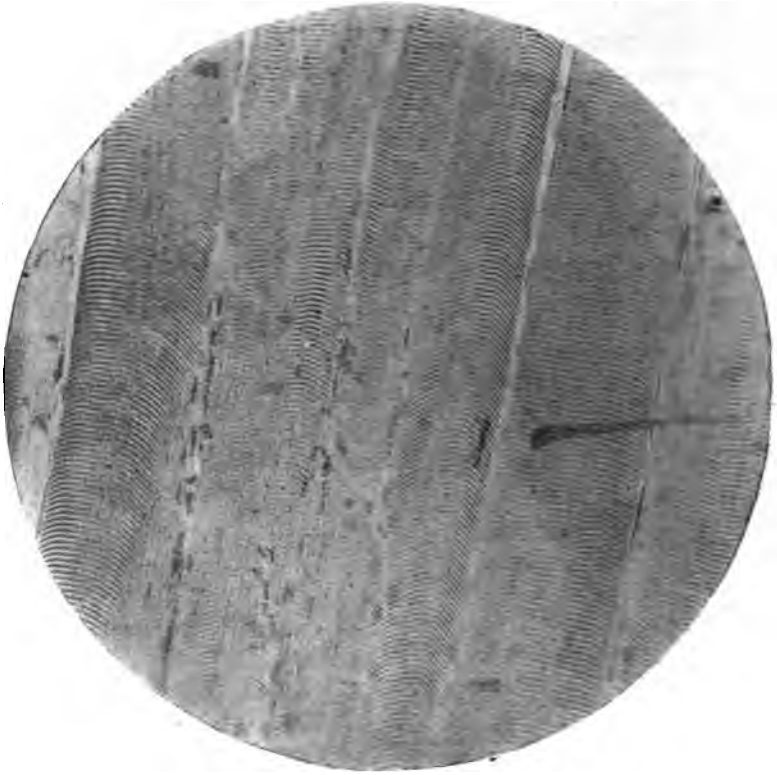

Fig. 4—Muscle showing transverse striations. Paramyoclonus multiplex.

They were twice or three times the size of normal fibers from corresponding muscle utilized as control preparations. Muscle fibers vary considerably in size even under normal conditions. In general, the larger the muscle the larger is the muscle fiber. In man the minimum diameter is given as 10 micromillimeters; the maximum as 100 micromillimeters. In the case under consideration, the larger proportion of the muscle fibers on transverse section averaged a diameter of 150 micromillimeters, many measuring 175 micromillimeters. This increase in size of the fibers was not universally distributed over the microscopic field. In round numbers, two thirds may be said to have presented this change. Many of those remaining were below the normal standard, so that the contrast was very evident.

Another peculiarity was the presence of sarcolemma nuclei between the sarcous elements. This is a peculiarity of the so-called red fibers of the lower vertebrates, a form of muscle which is more active, and capable of more prolonged contraction, than the white fibers which constitute human muscle, although such red fibers are scattered in very small number throughout the muscular system in man.

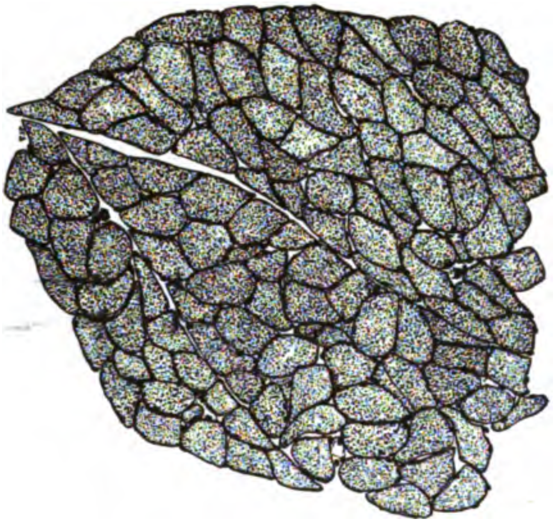

Fig. 5—Sartorius muscle from a case of chronic progressive chorea [control preparation]. Same enlargement as Fig. 3.

It is interesting and significant to observe that changes similar to those just mentioned have been described in cases of myotonia congenita (Erb,[35] Dejerine and Sottas[36]), the hypertrophic stage of the muscular dystrophies, and in those rare cases of true muscular hypertrophy, of which examples have been described by Friedreich,[37] Auerbach,[38] and Berger[39]. If such alterations in the size and nuclear constituents of the muscle fiber were simply the result of overaction, and secondary only to violent and prolonged muscular action, they should occur in long-standing spasmodic affections of cerebral origin. In one of the cases used as a control preparation in the present study, a chronic progressive chorea of fifteen years' duration, the muscles were found absolutely normal (Fig. 5). It is suggestive that in the muscular dystrophies and hypertrophies and in myotonia and in paramyoclonus multiplex, which represent obscure affections of the muscular and neuromuscular systems respectively, muscle changes essentially similar should be encountered. Hajos'[40] case of paramyoclonus multiplex with the electrical reactions of Thomsen's disease is of interest as furnishing clinical evidence of this relationship. We are far from understanding the true significance of the relations existing between the peripheral motor neurone and the muscle fiber, tissues having a different structure and derivation and yet intimately related; the fate of the one resting entirely upon the integrity of the other—the so-called *trophic function or influence of the anterior horn cells.*

In articular muscle atrophies, this trophic influence in some obscure way appears to undergo a reflex inhibition. In the Darkschewitz case the muscle fibers had suffered a reduction of one half their natural size.

As this trophic influence may under certain conditions be so perverted as to cause disintegration or atrophy of the muscle fibers, it is reasonable to assume the possibility of an increased trophic function with a resulting hypertrophy. If the Friedreich theory of the irritability of the anterior horn cells is accepted in explanation of the motor phenomena of paramyoclonus, these muscle changes may well represent a concomitant increase of the trophic function of these same cells, and this would constitute important objective evidence of the spinal origin of paramyoclonus multiplex.

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# Society Proceedings.

## NEW YORK NEUROLOGICAL SOCIETY.

March 3, 1903.

The President, Dr. Pearce Bailey, in the chair.

*Tabes Associated with Hemiplegia.*—Dr. Joseph Collins presented a man, forty-two years of age, a bar tender by occupation. When twenty-four years old he had had a chancre, for which treatment was given. A year later an iritis developed, and disappeared after two months' treatment. There was nothing further until eight years ago, when he was seized with an attack of vertigo, which was associated with an aphasia lasting several days. There was no paralysis. Four years later there was a similar attack associated with vomiting. Last August the man fell in an attack in which he was only partially unconscious. Examination showed a right-sided hemiplegia, and this had continued ever since. On coming to the hospital, there was found in addition a very marked atrophy of the right shoulder and an absence of both knee-jerks. The pupils were small and regular, and reacted to accommodation but not to light. There were incontinence of urine and manifest ataxia. The case was presented as one of tabes, the latter probably antedating the attack of thrombosis from which the hemiplegia originated. Coincident with the attack of cerebral thrombosis there must have been an obliteration of one of the cornual branches of the anterior spinal artery. In reality, this case presented three conditions: tabes, cerebral thrombosis and a destructive poliomyelitis of very limited extent. Tabes and hemiplegia were rarely associated.

Dr. J. Ramsey Hunt said that he had examined this case in the City Hospital. He had been doubtful about its being an anterior horn disease, and inclined to the opinion that there was a plexus lesion.

Dr. M. G. Schlapp said that in some of these cases of hemiplegia certain groups of muscles were markedly atrophied. The electrical examination should decide whether the case was of central or peripheral origin.

Dr. J. Fraenkel said that in only a few of the cases of tabes with hemiplegia had the knee-jerk returned, and this had only been temporary.

Dr. C. L. Dana said that he had had a short time ago under observation a man with tabes whom he had seen the very day of the occurrence of the stroke. There had not been any return of the knee-jerk, and he thought this could only occur in the early stage.

Dr. Collins said that there had been no suspicion of the existence of tabes before he had examined this man, and he had been deeply interested in the presence of the marked muscular atrophy.

*A Case of Major Hysteria* (?).—Dr. Collins then presented a woman, forty-four years of age, a cook by occupation. There was no history of alcoholism or of venereal disease. The present trouble began four years ago with an involuntary twitching of the hands and a loss of sensation in the hands and arms. This loss of sensation had gradually spread over the entire body. She had no pain, but felt tired and walked with difficulty. The gait was not strictly ataxic, but was rather shuffling and choreic. There was a peculiar condition of the left lower extremity, the lower third being shrunken without any hardening or thickening of the skin. Examination showed universal analgesia and more or less thermal anesthesia and analgesia. The knee-jerks and ankle jerks were exaggerated. There was slight disturbance of speech but not of intellection. She presented none of the ordinary stigmata of hysteria, and the color fields were of normal size. The woman had not improved under hospital treatment. According to the history, her father and sister were similarly affected, but the former lived to be over seventy years old. The case was presented without a positive diagnosis, because it did not seem to Dr. Collins to correspond to hysteria, syringomyelia or Huntington's chorea.

Dr. Schlapp was of the opinion that the case was one of hysteria, because the condition did not correspond to any known pathological lesion. The condition of the leg might have been brought about by a self-inflicted trauma. Two years ago he had presented to the Society a case of supposed atypical zoster, but subsequent investigation showed that the patient had produced the condition herself by means of carbolic acid.

Dr. R. H. Cunningham said he was reminded of a number of cases that he had had in a family in Richmond, Va., in 1893. Choreic symptoms developed between the ages of fifteen and thirty in the father, two sons and two daughters. One of the daughters had hemianesthesia. He would look upon the case just presented as one of adult chorea.

Dr. Collins said that he had been inclined to look upon the case as one of hysteria, yet this diagnosis presented many difficulties. After having observed the patient carefully for several months he was still more inclined to the diagnosis of major hysteria.

Dr. Pearce Bailey said that he thought the ataxic gait was due to the anesthesia, and that the case was one of major hysteria. With regard to the other case, there seemed to be no doubt that the atrophy of the shoulder was distinct from the tabes. A fall on the point of the shoulder was quite frequently the cause of a paralysis with just such an atrophy.

*A Case of Spinal Tumor.*—Dr. M. G. Schlapp presented a man, forty-four years of age, with a good previous history. About two years ago a peculiar twitching had been noticed in the muscles over the right shoulder, and about two months later he had begun to suffer pain at the root of the neck and extending into the trapezius muscle. After a time the right arm became weak, and subsequently the left side. This weakness was found to correspond to the muscles supplied by the anterior and posterior thoracic nerves on both sides. There was the Babinski symptom on each side. There were analgesia and diminished temperature sense on the left side. The diagnosis made was an extramedullary tumor involving the fifth and sixth cervical roots of the spinal cord, pressing upon the pyramidal tracts, and on Gowers' tracts, diminishing the pain sense and temperature sense. The tumor lay on the right side, and probably involved the motor roots on the other side. He thought an intramedullary tumor could be excluded. The man was to be operated on.

Dr. Pierce Bailey said he had seen this man last Fall, and at that time there was no sensory disturbance, and the pain dated back two years, but with intermissions. He had formed the opinion then that there was a degenerative lesion rather than a tumor.

*A Case of Cerebellar Tumor.*—Dr. J. Ramsey Hunt reported a case of cerebellar tumor with degenerations of the posterior columns. The subject of this report was a man of forty-six who had been admitted to Bellevue Hospital in July, 1902, with typical symptoms of cerebellar tumor, although the tumor could not be localized. The cerebellar symptoms had begun three months before, and the patient was under observation for three months. During these six months there was a progressive diminution of the knee-jerks and Achilles jerks on both sides. It was thought to be due to degenerations in the posterior columns of the cord, as had been described in connection with tumors in the posterior fossa of the skull. There was very marked choked disk, and the man's mental condition became one of apathy and somnolence. At the autopsy two small tumors were found, one springing from the dura mater in the median line, and encroaching somewhat upon the right frontal lobe, and the other tumor was in the left cerebellar hemisphere. Both tumors were medium-sized round-cell sarcomata. The spinal cord presented evidence of greatly increased intracranial pressure. Specimens of the spinal cord, both transverse and longitudinal, were prepared, and they apparently showed degenerations of the posterior columns, such as had been described in connection with tumor. The higher levels of the cord were most affected. Lissauer's column was spared, as was the rule. The fibers in Clarke's columns were also usually spared. These degenerations arose from the entrance of the posterior roots into the spinal cord. It was an anatomical fact, that where the posterior roots perforated the dura mater obliquely there was a constriction, and that here the sheath of Schwann was lost. At the same time there was a constriction of the myelin substance, and some of the fibers were deprived entirely of this substance. The speaker then briefly discussed the mechanical and toxic theories, and said that he presented the case as one favoring the mechanical theory.

*A Case of Cerebellar Tumor.*—Dr. L. Pierce Clark reported the following case: The subject was a man of thirty, who had had both gonorrhea and syphilis, and who used alcohol and tobacco to excess. The present illness began in October, 1901, with headache in the right occipital region. After a time he began to vomit in the mornings, and the right side became weak. Iodide of mercury had been given without benefit. On examination, there was choked disk in the right eye; the sixth nerve was bilaterally weak; the muscles of the right leg were stiff and painful; the head was held stiffly and bent slightly to the right side; all the deep reflexes were exaggerated, particularly on the right side; there was double Babinski; the gait was characteristically cerebellar. A diagnosis was made of a syphilitic tumor in the middle lobe of the cerebellum involving the peduncle. On admission to the Presbyterian Hospital in January, 1903, for operation, an X-ray examination showed the tumor. The man died rather suddenly, and the autopsy disclosed a tumor of the cerebellum. Between it and the fourth ventricle was a large cyst filled with slightly turbid serum. Apparently the growth was a syphilitic gumma that had undergone syphilitic and calcareous degeneration.

*A Case of Spindle-Cell Sarcoma of the Cerebral Dura.*—Dr. B. Onuf reported this case and presented a specimen. The subject of the report had been first seen on May 2, 1902, and at that time complained of a shooting pain in the left ear and in the teeth, and of attacks of dizziness. There was only slight perception of light; the gait was staggering, there were no marked changes in sensation or in the reflexes; there was typical choked disk. A diagnosis of cerebellar tumor was made. Subsequently the man developed slight paresis of the left facial nerve with fibrillary twitchings of the muscles in this region, and the hearing on the left side became greatly impaired. In walking, the man deviated to the right. He died in December, and at the autopsy a tumor was found at the base of the left cerebellar hemisphere. It was a spindle-cell sarcoma. This could have been enucleated had it been in a more accessible situation.

Dr. Schlapp said that about a year ago he had presented to the New York Pathological Society a case similar to the one presented this evening by Dr. Hunt showing degeneration of the spinal cord. The specimens from his case had been counterstained with acid rubin and the sheath of Schwann was stained. In all of the cases the degeneration extended to this sheath, especially in the cranial nerves, the trigeminus, the glossopharyngeus and the vagus nerves. He had expressed the opinion at that time that the sheath of Schwann might have some protective influence. He did not think the case tended to disprove the toxic theory. There were no sensory symptoms in the beginning, and probably the degeneration was at first confined to the fibers passing from the posterior columns into the gray matter. The poison appeared to be selective in its action, involving only the reflex fibers. The degeneration was particularly marked in the sensory nerves.

Dr. W. M. Leszynsky said that he had been interested in the fact that respiratory failure had preceded cardiac failure in Dr. Clark's case. One of his own patients had died upon the operating table just as the cerebellar abscess was reached. The heart continued to beat for eight or ten minutes after the failure of respiration. If degeneration existed in the posterior columns it must act chiefly on the third or fourth lumbar segment in order to produce loss of knee-jerks; how, then, would one explain those cases in which the knee-jerk was absent for a time, then became well marked, and was alternately present and absent?

*Muscle Tonus and Tendon Phenomena; Their Relationship and Interpretation.*—Drs. J. Fraenkel and Joseph Collins presented this paper, which was read by Dr. Fraenkel. It was based upon tonometric examinations of 230 patients. The authors stated that at the present day the view most generally held was that the tendon phenomena were expressions of muscle tonus. General clinical experience showed a definite relationship between tendon phenomena and the tonicity of muscles. Hypertonia, next to exaggeration of the tendon jerks, was a symptom of disease of the pyramidal tracts. The tonometer used in this investigation was the invention of the house physician of the Montefiore Hospital. A total of 554 registrations had been made. The 230 cases were distributed as follows: Apparently healthy, 32; cases of tabes, 25; of organic disease of the nervous system, 33; functional disease of the nervous system, 40; pulmonary disease, 70; heart disease, 12, and various other chronic affections, 18 cases. There were 71 registrations classed as hypertonia. It was found to be frequently, although not always, associated with exaggerated Achilles reflexes. Hypotonia was found to be frequently associated with loss or diminution of the tendon phenomena. There was a comparatively small number of neurogene hypotonias in proportion to the cases of general hypotonia. Neurogene hypotonia and absence of diminution of the tendon reflex were found frequently associated. Neurogene hypotonia with exaggeration of the tendon reflexes was present in nine per cent. It was concluded that hypotonia with exaggeration of the tendon reflexes was present in nine per cent. It was concluded that hypotonia produced by damage of the ascending tracts of the spinal cord was accompanied by loss or absence of the tendon phenomena in every instance. In the cases of hypertonia absent or diminished reflexes were present in six per cent of the registrations. In the cerebral group there was found a larger percentage of hypotonias than of hypertonias. In the spinal group 100 per cent gave exaggerated tendon reflex and 95 per cent showed hypertonia. It had been learned that whenever the neurogene tone was markedly increased or decreased the tendon jerks were increased or decreased correspondingly. The authors concluded that disease of the posterior tracts caused hypotonia, and disease of the pyramidal tracts caused hypertonia. There was a large group of cases giving tonicity and normal tendon phenomena.

Dr. G. L. Walton, of Boston, said that in the exhaustive discussion on reflexes and tonus opened by Crocq in 1901, the reader had reviewed the clinical and experimental evidence which tended to show that the reflexes generally corresponded to the tonicity. The exceptions to this rule he explained as illustrations of the fact that the centers for tonicity (at the cortex) and those for the deep reflexes (in the basilar region) were separate. This explanation was not directly discussed, though the limitation of these functions to the regions indicated was seriously questioned. Drs. Fraenkel and Collins had reinforced in a most convincing way the results of Crocq, though their explanation of the exceptional cases differed from his materially. The question was too complicated to be discussed in its entirety, especially in the absence of exact knowledge as to the seat of either the tonus or the reflexes, but such work as had been reported by Drs. Fraenkel and Collins furnished one of the definite steps by which one might gradually mount to a knowledge of this complex subject. We had been so long imbued with the notion that in man, as in the frog, the spinal cord was the center for reflexes and tonus that it was difficult to transfer our study of these functions to the brain. Even in case of disturbed reflex in cerebral disease we were still apt to think of the function of the brain as limited to the withdrawal, or the increase of influence upon the cord, through the pyramidal tract. For a long time the hypotonicity and loss of reflex sometimes found at the onset of cerebral hemorrhage was attributed to the shock conveyed to the cord. At the discussion already alluded to the disputants, while recognizing the importance of the cerebral influence, were by no means in accord with Crocq in limiting the reflex centers and the centers of tonicity to the brain. Dr. Walton said he had several times taken occasion to suggest that, instead of speaking of lower centers controlled, or inhibited, by higher centers, inhibited perhaps in their turn by still higher centers, we should recognize the combined action of all centers, cortical, basal and spinal, allotting to certain regions a predominating reflex function, liable to transference to a lower level on gradual withdrawal of upper level influence. According to this view each reflex movement might be regarded as a resultant of the activity of various loops of different lengths, connected with each other both laterally and vertically. The more he had observed the reflexes in cerebral disease the more he was inclined to accord to the cortex the chief rôle in the deep, as well as the superficial reflexes, and in the tonus. It was difficult, for example, to explain on any other basis the absence of deep and superficial reflexes in the following case: The patient was unconscious and hemiplegic, with a temperature of 105° F., and all reflexes absent up to the time of his death. The Kernig symptom was present, showing, probably, comparative hypertonicity of the hamstrings, but the extremities were otherwise obviously hypotonic. Autopsy showed pneumococcus meningitis of the convexity with encephalitic invasion of the cortex. In other cases of meningitis the reflexes were frequently preserved, but in one case of meningitis coming under his observation, in which both brain and cord were affected, during the stage of rigidity and obvious hypertonicity the reflexes were absent, but reappeared as relaxation set in, first on the side first relaxed. One must evidently look farther than to separation of the centers to explain such conflicting phenomena; in fact, different cases might require different explanations. Possibly it was partly a question of comparative tonicity. In the last case, for example, the hamstrings might have possessed hypertonicity so far in excess of the hypertonicity of the quadriceps femoris as to "snub" the knee-jerk. In long standing infantile cerebral hemiplegia with contracture the deep reflexes were often absent, though hypertonicity was apparent. Doubtless in some of these cases the contracture was in such position as to put the tendons on too great or too little stretch, thus mechanically preventing the reflex. The combination of exaggerated reflexes with hypotonicity was sometimes seen in Erb's syphilis of the cord, especially in the early stages. In a recent conversation with Dr. Courtney he had suggested that the affection about the fibers of the pyramidal tract was sufficient to impede the transmission of voluntary impulse and of tonicity from the cortex, but the reflex stimulus passed the more rapidly, as in neurasthenia, uninhibited by the volitional mechanism. Though not sure that he had thoroughly grasped the explanation of the readers on this point, Dr. Walton felt that they had proceeded along logical lines.

Dr. C. L. Dana thought the authors had established quite clearly the relations of hypertonia to the reflexes, and had given to the neurological world tangible working data. He had not fully understood the explanation of the effect of the brain on hypotonus and hypertonus. Personally, he made use of a working hypothesis which fitted in very well with the explanation given in this paper. In all the sudden insults to the brain occasioned by hemorrhages producing profound hemiplegia there was always absence of the deep reflexes on the paralyzed side, and with it no doubt hypotonia; whereas there was not this loss of reflexes on the non-paralyzed side. After a time this absence gave place to an increase of reflex. If, however, the hemorrhages were more posterior and involved the sensory sphere, there was more definite and more prolonged absence of reflexes.

Dr. B. Onuf referred to a case in which a tumor of the hip and shoulder centers was diagnosed, and the tumor removed, with the result that a very marked hypotonia developed in the paralyzed extremity after the operation, and was present even two months afterward.

Dr. Collins said that Dr. Fraenkel was satisfied with having established the relationship between tonus and reflexes, and they were both particularly gratified with Dr. Dana's statement that in his cases of cerebral hemiplegia he had observed that the farther posteriorly the lesion extended, either into the sensory cortex or the sensory representation of the capsule, so in proportion was the existence of hypertonia or the lateness with which it appeared. This clinical experience fortified very greatly the position taken in the paper.

Dr. Fraenkel said that he too had been delighted at the confirmation of their position by the very large clinical experience of Dr. Dana. If their observations were correct they should be of value in connection with diagnosis of pure lesions of the pyramidal tract associated with hypertonia and of the posterior tract with hypotonia.

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## PHILADELPHIA NEUROLOGICAL SOCIETY.

March 24, 1903.

The President, Dr. H. A. Hare, in the chair.

*A Case of Progressive Bulbar Palsy.*—This case was exhibited by Dr. T. H. Weisenberg. The patient had presented herself at the nervous dispensary of the Polyclinic Hospital three weeks previously with the statement that four weeks before coming to the clinic she had suddenly lost the power of speech. This she attributed to the wearing of false teeth. She protrudes the tongue in a straight line and moves it from side to side. There is a fine fibrillar tremor in the tongue. She can not pucker up the lips. There seems to be no involvement of the upper distribution of the seventh nerve. There is no apparent atrophy of the muscles of the tongue or mouth. She has typical bulbar speech. She swallows with difficulty. There is preservation of the pharyngeal reflex. The jaw-jerk is present. The knee-jerk is normal. The gait and station are normal. On laryngoscopic examination, no gross change is observed.

Dr. Charles W. Burr called attention to the fact that it was rare in a case of this kind to find such great involvement of the speech with such slight palsy of the lips as was present in this woman. As a rule in disease of the bulb when the speech becomes so bad as not to be understood there is marked palsy of the muscles of the tongue and lips.

Dr. William G. Spiller said that a curious feature about this case was that the loss of speech came on suddenly. This he thought was an error of observation on the part of the patient, although such a loss may occur suddenly, probably from hemorrhage. This he considered a case of true bulbar paralysis, not a case of pseudo-bulbar paralysis.

*A Case of Tuberculous Meningitis with Secondary Infection.*—This was reported by Drs. S. S. Kneass, W. F. Hendrickson and Joseph Sailer. The patient, a man aged twenty-nine years, was admitted to the Hospital of the University of Pennsylvania, March 15, 1902. His illness began with headache, nausea, vomiting, diarrhea and soreness and stiffness in the back. He had been treated for typhoid fever until March 8. He had gradually improved with the exception of the pain in the head. On March 12, in the third week of the disease, he became delirious.

Examination on admission showed moderate leucocytosis, diazo reaction negative, Widal reaction negative. On account of the cerebral symptoms, lumbar puncture was performed, and examination of the fluid showed the presence of a diplococcus. The patient was apathetic, but intelligent, answering properly, and did not complain of pain. Both pupils reacted normally. There was general hyperesthesia to all forms of sensation. Respiration was abdominal and the abdomen was retracted. There was a loud, short systolic murmur. Examination of the lungs was negative. The knee-jerks were absent, but there was no spasticity. The patient developed edema of the lungs, and on the second day after admission became comatose. Under the use of digitalis the edema of the lungs cleared up, but he died on the 19th.

Autopsy.—The heart was not enlarged,—no valvular lesion. The lungs were congested and edematous. The kidneys normal. The brain showed marked congestion, and at the base a puriform exudate into the meninges. Numerous tubercles were found on the meninges, and smears showed a large number of tubercle bacilli. The lungs presented a number of microscopic miliary tubercles. The meninges also showed marked infiltration of the lymphoid cells. The interesting feature of the case was the mistake in diagnosis based upon the presence of the *Micrococcus tetragenus* found in the spinal fluid. As far as the authors were aware this was the only case in which this had been found. The primary focus of the miliary tubercles was not found.

Dr. Alfred Gordon said that the most recent cytological studies of the cerebrospinal fluid threw considerable light upon the question of meningitis. These tend to show that lymphocytosis of the cerebrospinal fluid is a characteristic feature of an inflammatory condition of the meninges. It would be, therefore, interesting to know the result of the cytological examination in Dr. Sailer's case.

*Hemiplegia without Gross Lesions in a Case of Plumbism and Nephritis.*—This paper was read by Dr. Charles W. Burr. The patient was a man aged fifty-five years, admitted to the hospital February 3, 1903, in an unconscious condition. He had worked in the salts of lead. There was palsy of the right arm and leg and of the right side of the face. Both knee-jerks were fairly active. There was some extension of the great toe on stroking the sole of the foot on the paralyzed side. The man was stupid and delirious but not absolutely unconscious. On the second day, he became completely unconscious, and seventy-two hours after the onset of the paralysis, which had been sudden, he died. There was a distinct lead line on the gums, hyaline casts and a line of albumin were found in the urine. No gross lesion was found in the brain, and the only lesion discovered was chronic inflammation of the kidney. There was no arterial sclerosis in the brain or other part of the body.

*A Medullary Nucleus not as yet Described.*—A brief report of this was made by Dr. E. L. Mellus, of Baltimore.

Dr. Joseph Sailer thought that Dr. Mellus had proved the point for which he contended. What he had shown was certainly a nucleus. He congratulated the Society on the honor done it by Dr. Mellus in making the first report of the discovery to it.

Dr. William G. Spiller said that there was a distinct group of cells in the area referred to. There is also a similar group of cells in the human brain, although in the dog the cells are much larger. He raised the question whether this might not be a part of Dieters' nucleus. Whether or not it is to be considered a part of Dieters' nucleus, it is a distinct group of cells and has as much right to be considered a special nucleus as von Bechterew's nucleus which also may be regarded as a part of Dieters' nucleus. The determination of this question will rest with the discovery of the function of these cells.

*A Case of Complete Brachial Monoplegia.*—This case was presented by Dr. G. M. Dorrance. The patient was a man aged twenty-four years, who had come to the nervous clinic of the Polyclinic Hospital. In July, 1902, while working in a shaft an iron bucket fell, striking him on the left shoulder. He was rendered unconscious and remained in that condition for seven days. Since the accident he has not been able to use the left arm. The muscles have gradually wasted and the reactions of degeneration are present. The case was presented especially with reference to the propriety of operative procedure.

Dr. F. Savary Pearce said that he had presented a somewhat similar case about a year ago. There was degeneration-reaction throughout the brachial plexus distribution, and there seemed to be no help for the man, and amputation was finally done. Microscopic examination of the nerves of the brachial plexus showed widespread degeneration. It was Dr. Pearce's opinion that in a case of this kind, where there was so much degeneration and the member was useless, similar operations should be done. There was no delay in healing of the stump in the case Dr. Pearce reported, and the man was relieved of an incumbrance, much to his delight.

Dr. William G. Spiller referred to a case which he had seen in which Dr. Keen had exposed and resected the musculospinal nerve. A year and a half later there was almost complete return of power. In some cases there has been tearing and thickening of the nerves, and resection of the thickened portions and bringing the ends together has been followed by good results.

# Periscope.

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## ARCHIVES DE NEUROLOGIE

(No. 86, 1903, February.)

1. Contribution to the Study of "Folies" by Contagion. G. CARRIER.
2. On Obsessions and Impulsions (Episodic Syndromes in Degenerates). DR. SOUTZO, JR.

1. *Contribution to the Study of Folies by Contagion.*—The author states that "morbid contagion in mental troubles may be defined as the action of suggestion exercised by a diseased mind upon one or several sane minds, which reproduce it, each according to his individual disposition, either as a simple belief in the disordered idea or in insane manifestation of every form. This state created by contagion has been denominated in the nosological classification of folie, under the name of *folie à deux, delire à deux,* etc., expressions which we think too general and which we replace by *folie par contagion."* The author states that this definition has the advantage, first, of comprehending the numerous observations published on this subject, and second of being general; *folie à deux* being only a special effect of suggestion in the morbid psychical sphere; suggestion which acts in so many diverse forms in normal psychical life." I. The article proceeds with an extensive bibliography of the subject, beginning with Baillarger in 1857, who in that year first described *folie à deux,* and mentioning among others Oscar Woods, Jelly, W. Griffin and R. Dewey. II. In order that folie by contagion exist, there must be two principal pathogenic elements: contagion and morbid suggestibility or predisposition. "Morbid contagion is the *raison d'être* of all forms of folie by imposition, of folie by communication, of folie by transmission or by the simultaneous existence of insane phenomena." This was long denied by the authors, as Regis, etc., but is now fully established. Suggestion, the authors say, has three factors: imitative, persuasive and intermental action; this last factor being, according to M. Tarde, the conditions under which one mind is able to act upon another. III. The author reviews the determining causes of the development of folies by contagion. There are three groups of causes; the first accentuates the suggestive elements; the second addresses itself to predisposition, and the third to impression. In the first group are 1st, a long life in common and as intimate as possible; 2nd, habitual ascendency of the unsound upon him or them of sound mind, etc. In the second group: general wretched condition, illness, intoxications, debilitatng vices, etc., physiological states, like puberty, menstruation, pregnancy, accouchment, lactation, menopause. The third group: impressions produced by insane phenomena. IV. In all folie by contagion there are two elements: an active subject, contagious element; a passive subject, element receptive. "The active subject in general exhibits intellectual faculties more vigorous, a will and an energy more powerful than the passive subject: he is, in a word, more suggestive. His intellect, however, may not be superior, may even be inferior to that of the passive subject. the acuteness of the insane phenomena suffices to create the suggestive state. The passive subject is one whose intellectual faculties are enfeebled in various degrees by a predisposition whether hereditary or acquired; in a word, he is suggestible. He may reject the morbid impressions when the influence is withdrawn; or he may appropriate, develop

and transform them and give them a form quite personal. The author recognizes three clinical forms of folie: folie imposed, folie simultaneous and folie communicated. The author does not agree with the opinion of Marandon, who thinks that the passive subject never has hallucinations, and that he should not be classed as insane. He cites the cases of a young man and a young woman, in which the latter became the active subject of folie imposed. The young man had cohabited with the woman as his mistress for several years. She developed insanity of the alcoholic type, ideas of persecution, jealousy, hallucinations of hearing and sight, disorders of taste and smell. Her delusions led her to complain to the police, and her jealous threats frightened the young man. He consulted his physician, but under delusion said that the persecutions complained of by the woman were realities. The physician advised that he have the woman taken to an infirmary. He did this a few days thereafter. A month after the woman's admission, he became quite restored to mental health, and the woman, after four months of isolation and treatment, was discharged cured. The difference between folie imposed and folie communicated is that in the former there is merely a transmission of delusions, while in the latter the patient evolves delusions of his own, in addition to those imposed on him. The patients must be separated for their treatment. The author furnishes at considerable length two other cases under his observation, one where the patient was the active agent in the folie by contagion, and the other where the patient was the passive agent. The author concludes that passive subjects of folie imposed must of necessity be considered in a suggestive state and consequently in a medico-legal point of view irresponsible, so long as they are in that state.

2. *On Obsessions and Impulsions* (*Episodic Syndromes in the Degenerate*). Degeneracy betrays itself, says the author, by a certain number of physical signs generally grouped under the domination of stigmata. The external manifestations of morbid syndromes are obsessions, impulsions, inhibitions, perversions of instinct, various delusional troubles, all of which arise from disequilibration of cerebral departments. Obsession and impulsion have two elements: a center entering suddenly into function, by itself, without being solicited by the need of movement, and a momentary impotence of the will to escape the phenomenon which imposes itself. There is a rupture of the harmony of cerebral centers and exuberance of certain of them. We believe that we may affirm that volitional element has no action in the genesis of obsession. It has been claimed that obsession is a malady of the will. As we see it, says the author, all that occurs, occurs without exercise of the will, and if this last appears to intervene, it is in impulsion only, and in such a case it appears simply suspended, momentarily annihilated as a sequence of the struggle which it opposes against a very superior force, a force due to the *exaggeration* of one or several psychical centers in a state of excitement. The obsedant idea in the degenerate is born with the individual, remains a long time latent, until the moment when an appreciable cause makes it break forth; then increasing little by little it reaches a maximum and constitutes the morbid troubles called episodic syndromes. The author sets forth with much detail five cases of syndromic states: ideas of homicide, suicide, doubt, theft, perversions, etc. The first was of a single woman, thirty-three years of age, with conscious ideas impelling to homicide, suicide, with fears of all instruments capable of causing death, like knives, needles, pins, etc.; there was perversion of sexual instinct, also onanism. She entered a private hospital, "was submitted to two seances of hypnotism," *suggestion* gave some tranquility, and finally distractions, reading, and above all an alimentation sufficiently abundant, joined to daily moral treatment, improved her state, so that now she has only some abdominal troubles and headache. There were no signs of degeneracy, but clear indications of hysteria. The author remarks that the syndrome of impulsion, contrary to the claims of certain clinicians,

did not lead fatally to action. The second observation is upon the case of a woman of thirty-four, married at the age of nineteen, had 6 children and 2 miscarriages. One son at the age of 16, after typhoid fever, had committed suicide. This misfortune rendered her sad, but under medical care she recovered perfect health. Without any appreciable cause she once attempted suicide by poison, and twice left her home with the idea of suicide; once, thinking of her 5 children, she returned home, and the third time remained in a forest five days without eating, hoping to die by starvation. Admitted to the asylum she said: "I don't know, the idea of suicide came to me suddenly without any reasons. It is stronger than I." In this case the syndrome of suicide was the only one; its access was sudden, and there was no resistance on the patient's part. Such individuals are scarcely responsible for their crime. The third observation was of a woman of forty-one, married. There was a history of gross sexual perversion, onanism. She had impulses to break crockery, to kick at stones in the street, to give blows to persons who were passing beside her. She was for three years a janitress, and believed that in rendering her accounts to the manager she had paid over a sum less than she owed. From that moment she became sad and preoccupied and doubted whether she had not lost some banknotes. In the street she was impelled to rummage the pockets of people, believing that they contained banknotes. Taken to the hospital she saw banknotes everywhere; at sight of notes her face became red, she briskly recoiled and feared to touch them. By degrees, under the moral treatment of the physician, which had great influence with her, she became less anxious and at the end of six months entirely re-established. At the present time she is lucid and touches banknotes without emotion and recognizes the absurdity of her former ideas. In this case there is a co-existence of a certain number of obsessions and impulsions, which appear little by little during a period of five years and ends by a cure, which is the usual termination. Observation four is a case of impulsion to theft and suicide in a woman of forty-five. She was severely ill with typhoid fever, had chorea, hysterical crises. She was employed at a dressmaker's shop; at thirty-seven she took up this work again at Paris, but the handling of the silk enervated her greatly and brought on "crises" almost every week. A second and more severe attack of typhoid rendered her more dull, and at the same time arose the impulsion to theft. The impulsion appeared suddenly and was followed always by the act, resistance almost nil. The object of her theft was always silk, and by preferance red silk, and the act was accompanied by sexual orgasm. For a theft at the Bon Marché she was condemned to six months in prison. (A note says that she was reported to have undergone sixteen sentences for numerous thefts in shops.) She was brought to the asylum after having thrown herself from a window on the second floor of the house where she lived; she remained there three months. After an hysterical crisis, she attempted to throw herself before a railroad train, but was prevented by her daughter. The next day, without being completely dressed, she took a cab and went to a shop, where she stole a package of silk. She was arrested and taken to the Infirmary. At the hospital she seems debilitated, rubs her hands constantly, is restless, weeps at intervals. The author considers this an example of the possible co-existence of mental troubles, syndromic and nervous. It shows hereditary degeneracy, re-enforced by fibrile troubles. Observation 5: Case of a married woman, who was brought to St. Anne in 1902 for the third time. Her first morbid episode occurred thirteen years ago, when she had fear of needles, glass and fire. She could not see a candle lighted without fear of seeing it kindle a fire. In buying an object, after paying for it, she doubted she had not paid less than the sum due. She became anxious and frequently wept and was taken to the asylum for the first time. She was discharged after three months completely cured. After an interval of 11 years, she was seized with depression, multiple hallucinations of hearing, sight, ideas of culpability, of persecution, and some of her former ideas of doubt. She left the asylum in 30 days, again entirely cured. She passed two months in the country, returned to Paris in October, 1901, and by degrees new ideas entered her brain. She became very religious, went to church every day, though before she did not even go on Sunday. She told her husband that she was poisoned, etc. Her troubles became aggravated, and in February, 1902, she was again admitted at St. Anne. Mystic ideas predominated: she complained of suffocation, palpitation, night visions, etc. After three days she became lucid, all had disappeared; she only felt general fatigue. This clinic shows us three forms of trouble; all belong to the same base—degeneracy. There were not three maladies belonging to three periods, for then there would be three different conditions, one attached to each of the morbid periods. The author concludes: "Finally from this exposition of what the clinic has shown us, we think that we should state some particulars concerning a certain number of important syndromes. The group of obsessions and impulsions to homicide and suicide present some special characteristics. The obsessional idea is more tenacious, the struggle on the part of the subject is increased by the will, which opposes itself with a greater energy; in fine, the moral torture reaches a maximum. In what concerns the performance of the act, we should add, and with much justice, that Magnan distinguishes two varieties of impulsions. In the first variety, the onset is sudden, automatic, with no anterior resistance or reflection in the subject (see second case above); in the second variety, the person holds himself for a long time as master of the obsedant idea, or rather at the same instant that he sees himself on the point of yielding, he has recourse to the aid of a member of his family or other such help; 'he gets the start of his environment' (*il prévient son entourage*), according to the expression of Magnan (see second case above). In fine, in the medico-legal point of view persons, who commit crimes under the influence of these morbid impulsions are not responsible, and such persons, as well as the criminal-born of Lombroso, are only, as we have seen, the unbalanced, belonging to the great family of degenerates. The second syndrome, the folie of doubt, demands also some comment, because of the different interpretations which it has received from alienists. Falret, Sr., who first described folie of doubt in an admirable manner, commits the great fault of isolating it too much. He makes of the syndrome a distinct malady of progressive character and a definite prognosis: incurability. Le Grand du Saule, in his monograph, associates it with the delirium of touch; to his mind this intimate association exists of necessity, and the appearance of the delirium of touch marks the debut of a second period in the morbid entity, formed by this association. There are at this time among the clinicians a certain number who still entertain these opinions. As for Magnan and ourselves, we are far from looking upon the folie of doubt under such an aspect; it constitutes merely a syndrome, which may exist in the clinic, either by itself or at times associated with the delirium of touch, and that only by preference, for we may meet it also with other syndromic states. The prognosis is habitually favorable, but on the cardinal condition of never neglecting the patient, of having him each day under one's eye, and of instituting for him a continuous medical and moral treatment. This view is, besides, justified by all our observations. The other syndromes set forth in our cases give a precise idea of the general characteristics indicated at the commencement of this essay. Briefly, in conclusion, we think that we may affirm that this study shows us once more that episodic syndromes, varied as they may be, belong to the same foundation, mental degeneracy; on the other hand, it permits us to place upon the solid foundation of the clinic, in the medico-legal appreciation, faulty and criminal acts committed by individuals belonging to the great class of degenerates." MR. RICHARDS (Amityville).

### ARCHIVES D'EDLECTRICITE MEDICALE

(No. 120, 1902, December.)

1. An Adjustable Inductance for High Frequency Resonators. H. Guilleminot.
2. On the Use of an Electrolyte in Parallel to the Primary Current of a Coil for the Production of X Rays and High Frequency Currents. Bordier and Nogier.
3. Anesthesia by the High-frequency Brush Discharge in the Ablation of Protruding Hemorrhoids and Hemorrhoidal Condylomata. Billinkin.
4. Electric Cerebral Inhibition in Man. Stéphane Leduc.
5. Measuring the Diameters and the Area of the Heart upon the Radioscopic Screen without Drawing. A New Arrangement Adapted to Any Screen. H. Guilleminot.
6. Death and Accidents by Industrial Electric Currents. F. Battelli.
7. Congress at Berne. Exhibition of Apparatus for Medical Electricity and for the Production of X Rays.

1. *Inductance for Resonators.*—The author describes his method of more accurately tuning resonators by the introduction of an adjustable self-induction coil in the primary oscillating circuit, a device that was employed by Tesla about 13 years ago.

2. *Electrolytes in the Primary Circuit of Induction Coils.*—The writers recommend shunting the terminals of the primary by a small cell composed of lead plates submerged in very dilute sulphuric acid. Under certain conditions of current the relative spark length of the coil is stated to be considerably increased.

3. *Electrical Anesthesia in the Excision of Hemorrhoids, etc.*—In this paper the author fully describes his mode of using a high-frequency brush discharge applied to the anus to produce anesthesia and constriction of the neighboring blood vessels. With suitable apparatus properly used a painless and practically bloodless operation can be performed.

4. *Cerebral Inhibition.*—A description of a method of producing partial inhibition of the sensori-motor cerebral centers by causing a rapidly interrupted electric current to traverse the brain and spinal cord. The negative electrode is placed upon the forehead and the positive on the lumbar spine. A 50-volt generator of low internal resistance is used to supply the current in conjunction with an adjustable shunt rheostat, while the current is interrupted by means of a Gaiffe mercury interrupter. The sensations of the subjects, Prof. Malherbe and Alfred Rouxeau, upon whom the investigations were made, were as follows: "When the current was at a maximum we could still hear as in a dream what was said; we were conscious of our inability to move and to communicate with our colleagues; we perceived the touches, the pinching, and the pricks upon our arms, but the sensations were dulled like those from a limb fast asleep, etc." No disagreeable after-effect resulted.

5. *Measurement of the Heart Area, etc.*—A short desciption of the author's new disposition of his previously-described frame with horizontal wires and sliding balls, designed to measure the diameters and the area of the heart, projected orthogonally upon the fluorescent screen. In its new form the frame is placed on the fluorescent side of the screen, and the measurements are read distinctly from the accompanying centimeter scale on glass.

6. *Death by Electric Currents.*—A lengthy and complete paper which should be read in full to be appreciated. The paper is an extensive summary of Prof. Prevost and Dr. Battelli's investigations upon this important subject, and also contains some very interesting remarks upon the electrocution of criminals in this country. Briefly stated, the author finds that low-tension continuous or alternating currents kill by causing cardiac

fibrillation; high tension alternating currents at 1,200 volts or more kill on the contrary by inhibiting the respiratory center. Alternating currents of from 240 volts to 600 volts produce in dogs cardiac fibrillation and often permanent arrest of respiration. High-tension currents, according to the author, do not produce cardiac fibrillation, but possess the property of causing a fibrillating heart to beat coordinately after it has been subjected to a previously applied low tension current. Upon the subject of criminal electrocution the author says: "With the introduction of the new method employed in electrocutions (a high-tension current followed by a low-tension current), the inhibition of the nerve centers is followed by cardiac paralysis. Thus burns are avoided, and the signs of life cease most rapidly. Electrocution, as all other methods of capital punishment, is a disgrace to our civilization, but it is the least repugnant because consciousness is immediately lost and the condemned is not disfigured, nor is the execution bloody."

(No. 121, 1903, January.)

1. Measuring the Actinic Power of the Radiants Employed in Phototherapy. A New Actinometer. Bordier and Nogier.
2. On Intra-rachidian Stimulation in Man as a Therapeutic Procedure. J. Bergonié.
3. On the Maximum Power of Currents Employed in Medicine and the Way to Obtain Them. H. Guilleminot.
4. Electrotherapy and Orthopedics. E. Albert-Weil.
5. The Present State of Electrodiagnostics. Ludwig Mann
6. A Five-Cell Electric Bath or Perineal Bath. Felix Allard.

1. *Measurement of Actinic Power in Phototherapy.*—As the authors consider it highly advisable to gain some idea of the intensity of the violet and ultra-violet rays to which the subjects are subjected in phototherapy, they have devised a special form of actinometer by which it is claimed that not only the relative intensity of violet rays can be accurately measured but also the intensity of X rays.

2. *Intra-rachidian Electrical Stimulation.* — A paper describing the effects of faradic excitation of the cord and nerve roots in three patients in whom the lumbar puncture was performed several times. The patients apparently experienced no pain and, luckily, experienced no evil after-effects.

3. *Maximum Intensity of Currents in Medicine.*—A paper based upon mathematical and theoretical considerations from which the author concludes as follows: (1) If a generator with unchangeable constants is employed in conjunction with a variable external resistance, the maximum intensity of current will be obtained by making the external $R$. equal to the internal $r$. (2) If the resistance of the external circuit is unvariable and several generators are employed, $q$ elements in parallel must be united into $t$ groups in series, so that $\frac{t}{q}=\frac{R}{r}$, in order to obtain the maximum current.

4. *Elcctrotherapy and Orthopedics.*—This paper is merely a brief sketch written to show that the use of electricity exerts considerable benefit in orthopedics, "but one must not forget that its value depends upon the manner of its application." The writer fears from his experience that the use of the galvanic current in atrophic paralyses in children is inapplicable, owing to the pain produced by the electrodes.

5. *Electrodiagnosis.*—A long summary discussing the merits and demerits of the various commonly employed methods used in electrodiagnosis, which is unsuitable for abstraction.

6. *Electric Perineal Bath.*—A description of an apparatus consisting of five vessels suitably connected to a switch board, so that one of special shape, in which the patient sits, serves as one electrode while the remaining four in which the hands and feet are inserted, form the other electrode.

(No. 122, 1903, February.)

1. On the Electric Treatment of Basedow's Disease. Th. Guilloz.
2. How to Understand the Contra-indications to the Electric Treatment of Uterine Fibromyomata. M. Laquerrière.
3. The Treatment of Gonorrheal Arthritis in the Acute Inflammatory Stage by Strong Continuous Currents. Louis Delherm.
4. The Action of Ozone Upon the Bacillus and the Toxine of Diphtheria. F. Arlving and M. Tronde.

1. *Treatment of Basedow's Disease.* The author reports two cases of Basedow's disease considerably ameliorated by the electro-cataphoretic method of treatment, in which iodine was introduced into the tissues by a strong continuous current.

2. *Treatment of Fibromyoma.* After discussing the various varieties of electric treatment and specifying those forms of this trouble to which the different varieties of current are most applicable, the author gives a number of absolute and relative contra-indications which should be read in the original to be appreciated.

3. *Treatment of Gonorrheal Arthritis by the Continuous Current.* The author advises that in cases of hydarthrosis, or gonorrheal arthritis a galvanic current of from 20 to 60 m. a. be applied as soon as possible once or twice a day. Large kaolin electrodes are to be used.

4. *Action of Ozone on the Diphtheria Bacillus.*—These authors find that by exposing cultures of Loeffler's bacillus to ozonized air (.025 gram to 1 liter the effect on the bacilli is as follows: (1) Ozone cannot kill the bacilli in a nutritive liquid. (2) It can attenuate their growth for 48 hours, although the proliferation after this period is less marked than in ordinary cultures. (3) It diminishes the virulence of the bacilli so that inoculated guinea pigs survive, and in those that die only lesions at site of the inoculation are found. When the toxin was exposed to 200 liters of the above-mentioned ozone mixture, the animals survived a 1 cc. injection, although the control animals died in 24 hours from a dose of 0.5 cc. R. H. Cunningham.

## JOURNAL DE NEUROLOGIE

(Vol. 7, 1902, No. 21, November 5.)

1. Sensorial Excitations as a Cause of Epileptic Seizures.. Ch. Féré.
2. Occipito-Cerebellar Syndrome. L. Marchand.
3. Exophthalmic Goiter. Treatment by Thymus. A. Bienfait.

1. *Sensory Excitation in Epilepsy.*—The author emphasizes the fact that sensorial as well as sensory excitations may provoke epileptic attacks, and relates the following cases: (1) In an idiot of eighteen years, subject to both diurnal and nocturnal attacks of grand mal, the diurnal seizures were found constantly to follow sensorial excitations. Sudden noises, sudden variations in intensity of light, a current of air, and even a strong odor sufficed to bring on a fit. (2) In a man of sixty-five years of age, epileptic from his youth, and having two or three fits monthly, loud noises and sudden changes in light intensity would often bring on an attack. The author saw a typical grand mal seizure produced in this case by a sudden blow on the table behind the patient. The lesson taught is that in epileptics careful investigation into the exciting causes of their attacks should be made, and attention should be paid to any idiosyncrasies in this particular which they may present.

2. *Occipito-cerebellar Syndrome.*—The author describes the case of a woman of forty-two years of age, who had had occasional attacks of epilepsy for five years. Seized suddenly with headache, diarrhea and vomiting, she speedily developed loss of vision, going on to nearly complete blindness, staggering gait, Romberg's symptom and exaggerated reflexes.

The ophthalmoscopic examination showed double pupillary stasis. She also had several fits. After eight days the gastrointestinal symptoms improved, but the next day she went into status epilepticus. Learning at this time that her husband had been syphilitic, the author began intramuscular injections of biniodide of mercury and iodide of potassium. She commenced to improve, and in about two months had regained her vision, and the headache and motor symptoms had disappeared, though the epileptiform attacks persisted. The author thinks that there is no doubt about the syphilitic origin of the symptoms, and diagnoses a gumma at about the insertion of the falx cerebri into the tentorium.

3. *Exophthalmic Goiter, Treatment by Thymus.*—The author discusses the pathogeny of Basedow's disease, and narrates some experiments of his own upon rabbits, in which incisions into the medulla about and into the restiform bodies produced some of the symptoms of this disease. That these symptoms probably arise from this region he thinks is shown by the results of experiment and by the finding there of lesions in a certain number of cases of Basedow's disease. Certain other maladies, notably tabes, which are progressive, have occasionally been found to cause some of the symptoms of Basedow's disease when they extend to the medulla. These symptoms, again, may arise reflexly or be neurotic in origin; and, lastly, the center in question may be susceptible to the action of certain poisons. The author thinks that the thyroid and thymus glands may in some way be complimentary in action, hence suggests the use of extract of the latter, and gives a report of a case in which he used this remedy with some, though not very positive benefit.

(Vol. 7, 1902, No. 22, November 20.)

1. Dementia Præcox. F. Meeus.

1. Upon the basis of his observations in 47 cases, the author discusses the etiology of, and attempts to construct a symptomatology for, dementia præcox. The disease, he thinks, is not limited to early life, but may arise at a later period, and should be considered as a special form of mental trouble, with characteristic mental and motor symptoms, proceeding certainly to dementia. The characteristic negativism, stereotypy, etc., he regards as due to disturbance of association, which prevents regular and orderly discharge of impulses, resulting in inhibition, broken by sudden and irregular or ill-timed explosions. The term dementia præcox he regards as too indefinite, and suggests that it be replaced by that of "hebephreno-catatonic dementia."

(Vol. 7, 1902, No. 23, December 5.)

1. Theory of Reflexes Cutaneous and Tendinous. C. Heldenbergh.
2. Congenital Facial Diplegia. O. Decroly.

1. *Reflexes.*—A discussion of the physiology and pathology of reflexes, which embodies no original investigations, and does not lend itself readily to review.

2. *Congenital Facial Diplegia with Ocular Paralysis and Trouble with Deglutition.*—Report of a case of this kind in an infant of seven weeks old. The author is inclined to think it due to a lesion of the bulbar nuclei.

(Vol. 7, 1902, No. 24, December 20.)

1. The Intracellular Network of Golgi in Nerve Cells. S. Soukhanoff.
2. Chronic Poliomyelitis. De Bray.

1. *Intercellular Network.*—A review of the findings of Golgi and Veratti, with a description of the intracellular network as demonstrated by the author himself, especially in the cells of the sympathetic ganglia, by the method of chrome-silver impregnation of Veratti. The author does not regard this intercellular network as conductive in function, but rather as analogous to the intracellular canaliculi.

2. *Chronic Poliomyelitis.*—The author describes the case of a man sixty years old, who subsequent to a fall developed atrophy of the hand

muscles of the Duchenne-Aran type, with some kyphosis in the upper dorsal region. From a consideration of the situation of the special nuclei of the hand muscles, in the lateral region of the anterior horn, and their blood supply differing from that of the other cells of the region, he draws the inference that in this case there was a vascular lesion produced at the time of the injury, and the cells of the nucleus in question atrophied in consequence, while the other cell groups in that part of the cord escaped.

**Allen** (Trenton).

### NEUROLOGISCHE CENTRALBLATT

(Vol. 22, 1903, No. 1, January 1.)

1. A Contribution to the Feeling of Consciousness. A. **Pick.**
2. Concerning Hysterical Confusion and Mental Aphasia. A. **Westphal.**
3. Concerning a Developmental Defect of the Nose (An Undescribed Stigma of Degeneration). H. **Gudden.**

1. *Feeling of Consciousness.*—Pick considers in this paper the bekanntheits Gefühl as a condition in hysteria, sexual neurasthenia, epilepsy, etc., and discusses the psychology of the conditions.

2. *Hysterical Confusion.*—Westphal reports a case of hysteria with a confusional condition in which the patient cannot answer questions, and when she did answer them the simplest questions were wrongly interpreted or wrong answers were given. The question as to whether true clonus is present in hysteria, is, according to the author, determined positively in this case.

3. *Nasal Stigma of Degeneration.*—Gudden pictures a patient with a defective development of the cartilage and tissue of the alæ of the nose and considers it a stigma of degeneration.

Vol. 22, No. 2, 1903, January 16.)

1. Concerning Disturbances of Sideward Movements (Flank Gait) in Hemiplegics. v. Schüller.
2. Hedonal and the Nervous System. v. **Lampsakow.**
3. Autogenous Nerve Regeneration. A. **Bethe.**
4. Answer to No. 3. E. **Münzer.**
5. Hysterical Confusion and Mental Aphasia. A. **Westphal.**

1. *Concerning Disturbances of Sideward Movements (Flank Gait) in Hemiplegics.*—The author says this is a new differential symptom for the diagnosis of organic hemiplegia from functional hemiplegia. It depends on the fact that a hemiplegic patient can walk sideways towards his palsied side with little or no difficulty, but on account of the spasticity and lengthening of the palsied leg, he has considerable difficulty in walking sidewise in the opposite direction, and must needs drag the paralyzed leg.

2. *Concerning the Action of Hedonal upon the Animal Economy.*—V. Lampsakow gives the results of his psychological study of a new hypnotic, hedonal. He finds that it is an active and harmless hypnotic: much stronger than urethan, without any disturbance of the heart or respiratory organs; that it can be given preparatory to the administration of chloroform to facilitate narcosis; that it is more valuable as a hypnotic in anemic weak patients than chloral, and that it may be given by mouth or rectum, but cannot be administered hypodermically.

3. *Autogenous Nerve Regeneration.*—A. Bethe presents a controversial article with Münzer concerning statements of the latter in a previous article in this Centralblatt.

4. *Answer to No. 3 (Bethe) by E. Münzer.*

5. *Concerning Hysterical Confusion and Mental Aphasia.*—Westphal renders a medico-legal contribution to disturbances of memory in functional conditions and malingering of mental disturbances.

**McCarthy** (Philadelphia).

### ALLGEMEINE ZEITSCHRIFT FUR PSYCHIATRIE.

(Vol. LX, 1903, Heft. 1 and 2.)

1. On Ganser's Symptom. LÜCKE.
2. Induced Insanity. F. WITTE.
3. Cases of Simulation. BOLTE.
4. Graudenz Penal Institution. SANDE.
5. Contribution to Study of Paranoia. H. SCHNEIDER.
6. Conception of Hypochondria. A. PICK.
7. Exhibitionists before the Law. G. BURGL.
8. Asylum at Strelitz. SERGER.

1. *On Ganser's Symptom.* Although not previously unknown, Ganser in 1897, called special attention to a peculiar condition in hysterical subjects, characterized especially by confusion, childish bearing and an apparent inability to answer simple questions as to names, dates, and other ordinary matters, or to recognize persons or objects formerly well known. Common among those who have come into conflict with the law, the conduct of these persons under examination may readily arouse a suspicion of simulation. In consequence of the presence of other definite symptoms of hysteria, however, Ganser thinks the manifestation a genuine symptom-complex, and in this he is supported by other observers. The author discusses this symptom and gives the histories of four illustrative cases. The matter he considers important on account of its medico-legal bearing, as the symptom is especially common in prisoners with hysterical mental disturbances. The diagnosis between this symptom and simulation upon the part of the patient, is to be made upon the presence of hysterical stigmata, periodical attacks of anxiety, confusing of persons, and terrifying hallucinations, together with a characteristic stupid and at the same time tense expression of countenance, and generally confused appearance of the patient. The symptom has been described by Nissl as occurring in katatonia, but the author regards it as being due in these cases to a hysterical groundwork.

2. *Induced Insanity.*—The histories of two cases, one that of a man of 59 years, presenting a typical example of paranoia with expansive and persecutory delusions, the other that of a woman of 51 years, of neuropathic constitution and heredity, and addicted to the abuse of alcohol, the housekeeper of the first patient. The second patient, in consequence of the troubles of her employer, being left without resources, began to drink more heavily than usual, and developed a psychosis of strongly alcoholic character, with hallucinations and delusions of persecution. Interned in the same asylum as her employer, the picture changed to one of paranoia with expansive and persecutory delusions, almost identical in character with those of the former. The author thinks that the influence of the stronger personality of the man, acting for years upon that of the weaker and less educated woman, already predisposed through neuropathic constitution and alcoholic indulgence, is responsible for the clinical developments in the case.

3. *Some Cases of Simulation.*—Clinical histories of six cases of this character. Not suitable for abstraction.

4. *The New Insane Department of the Graudenz Penal Institution.*—Not suitable for abstraction.

5. *A Contribution to the Study of Paranoia.*—A clinical history extending over the thirty-six years of life of a typical paranoiac, with a lengthy discussion.

6. *On the Conception of Hypochondria.*—The author thinks that hypochondriacal delusions are often built up upon a material basis. To illustrate his views he gives histories of the cases of two middle aged women in each of whom gonorrheal trouble of the genital organs led to depression with persistent hypochondriacal delusions. One case was improved by local

treatment, but in the other the morbid ideas persisted in spite of subsidence of the genital disease.

7. *Exhibitionists before the Law.*—As adviser to the court the author has had occasion within four years to examine twenty-five persons accused of exposing their genital organs in public. He thinks that in the great majority of cases this act is a manifestation of mental disorder. Each case he thinks should be considered under the following heads: (1) Is the act due to mental weakness? (2) To uncontrollable impulse? (3) Is it the result of an imperative conception ("Zwangshandlung")? (4) An accidental occurrence? (5) Due to carelessness? (6) Is it the voluntary act of a mentally normal individual?

Under 1 he includes both congenital and acquired mental weakness. In these cases the act may be due to lack of ethical feeling, and of inhibitory contra-conceptions. It is observed in imbeciles of various grades, in paralytic and senile dementia, in epileptic delirium, after trauma, and in the pathological reaction to alcohol common in epileptics and neurotic subjects.

(2) The cases due to uncontrollable impulse may result from sudden vivid hallucinations or delusions, and sometimes coming on from time to time appear to be a manifestation of periodical insanity ("periodical sexual psychopathy").

(3) The cases in which the act is the result of imperative conceptions, the author separates from those due to impulse. The presence of an imperative conception alone does not necessarily make the patient legally irresponsible, but it may be accompanied by an effort of such intensity as to cause an obnubilation of consciousness, in which case self-control is lost and mental irresponsibility may ensue.

(4) Exhibition may occur accidentally through forgetfulness about adjusting their clothing upon the part of weak-minded or mentally confused individuals.

(5) Especially in old and feeble-minded persons there may be such carelessness as urinating or adjusting a truss in the street.

(6) Lastly, exhibition may be deliberately practiced, generally under great sexual excitement, by mentally normal individuals, usually upon meeting one of the opposite sex in a lonely place. In each case an inquiry into all the circumstances of the case attending the misdeed should be made, and if possible the statement of the patient in his own language should be secured. By so doing the physician will often be able to detect gaps in the memory which indicate the presence at the time of some disturbance of consciousness (epilepsy?). Each case should be judged upon its merits. A history of repeated exhibitions, especially in a very public place before numerous witnesses, points almost unerringly to mental aberration.

8. *The Asylum at Strelitz.*—Description of a newly erected building for 180 patients. Unsuitable for abstraction.

ALLEN (Trenton).

# Book Reviews.

INTERNAL SECRETIONS AND THE PRINCIPLES OF MEDICINE. By CHARLES E. DE M. SAJOUS, M.D., Vol. I, Illustrated. F. A. Davis Co., 1903.

The first volume of this remarkable work contains eight hundred pages almost exclusively solid reading matter, the illustrations being chiefly upon plates. It is an attempt to arrive at new theories and principles regarding certain obscure points in physiology, by a careful study of the observations and experiments of others. While it has generally been the practice of the world to accord the highest praise to the original investigator, it must not be forgotten that the man who organizes the mass of facts collected by others, puts them in an available form, and deduces from them new ideas regarding their essential relations, may contribute more to the actual advancement of knowledge than the accumulator. It must not be forgotten that the physicist, Young, takes his rank among the world's great men although his actual laboratory work in physics was comparatively insignificant; that Herbert Spencer has done much to mould English thought in biology, although he was not a great original investigator. To those who can accomplish both—that is, collect the facts and analyze them, as did Darwin—highest rank in science is ever accorded. Dr. Sajous is the analyst. He has made, he believes, some valuable discoveries regarding the rôle of the adrenals and the pituitary body. Briefly stated, his theory is that the adrenal secretion poured into the blood endows the hemoglobin and the plasma with an affinity for oxygen, that the plasma carrying the oxygen then circulates among the tissues and accomplishes the internal respiration. This function gives the internal secretion of the adrenals an overwhelming importance. The adrenal glands are controlled by the anterior portion of the pituitary body, and the functional efficiency of this is in turn sustained by the secretion of the thyroid gland.

These views are sufficiently novel; the question arises: What claim have they to our credence? The first chapter deals with the physiology of the adrenal glands. To us the conclusions in many cases appear to be strained. The proof offered is inadequate. The experimental material is obviously carefully selected, but even then does not suffice to confirm the deductions. Take, for example, the section on the physiological effects of poisons, in which the facts are stated by the investigators entirely without reference to the adrenal glands, and yet changes are explained by Sajous with reference to these organs. Take, for example. the relation of the adrenal glands to chlorosis: the conclusions must appear to any clinician to be forced. Of course the reviewer must guard against too general condemnation of any strikingly revolutionary hypothesis, and it is impossible to give an adequate analysis of the book in the brief space allotted to this review. There is one point, however, to which we feel that attention should be called, that is the statement that the posterior pituitary body is the chief center of the nervous system to which the afferent impulses go and from which the efferent impulses arise, so that it practically governs the brain. To one who has studied the histology of this organ it seems incredible that a mass so scantily furnished with cells or nerve fibers could possibly accomplish all that is ascribed to it. If its embryology is studied it appears more than reasonable to assume that it, like the pineal gland, is a relic of a former organ, and comparative anatomy lends color to this view. What then, has Dr. Sajous to offer against it? Very little indeed: Its position, which we admit is central enough; the fact that injections of its substance are said to increase the blood pressure; and the fact that Berkeley's investigations have shown that it actually does contain parts of neurones.

Perhaps we have been rather harsh with this book, but we feel that Dr. Sajous has not made out an entirely good case for the plaintiff. Nevertheless, it is well written, indicates a vast amount of industry, and is exceedingly suggestive. The publishers have done their part well.

SAILER.

A SYSTEM OF PHYSIOLOGIC THERAPEUTICS. Edited by SOLOMON SOLIS-COHEN, A.M., M.D., Senior Assistant Professor of Clinical Medicine in Jefferson Medical College, etc. VOL. V, PROPHYLAXIS, PERSONAL HYGIENE, CIVIC HYGIENE, CARE OF THE SICK, by JOSEPH MCFARLAND, M.D., Professor of Pathology, Medico-Chirurgical College, Philadelphia; HENRY LEFFMANN, M.D., Professor of Chemistry in the Woman's Medical College, Philadelphia; ALBERT ABRAMS, A.M., M.D., formerly Professor of Pathology Cooper Medical College, San Francisco, and W. WAYNE BABCOCK, M.D., Lecturer on Pathology and Bacteriology, Medico-Chirurgical College, Philadelphia. Illustrated. VOL. VI, DIETOTHERAPY and FOOD IN HEALTH, by NATHAN S. DAVIS, JR., A.M., M.D., Professor of the Principles and Practice of Medicine in Northwestern University Medical School; Physician to Mercy Hospital and Wesley Hospital, Chicago. Philadelphia, P. Blakiston's Son & Company.

In the preface of Volume V of this series we read, "The following pages contain an epitome of what is essentially the natural history of medicine.' Such is the broad scope adopted by the editor, who takes a special pleasure in this volume "because of his success in having the subject of prophylaxis brought into close relation with that of treatment at so many points. The artificial conditions of civilization, the diversity, complexity and strenuosity of the activities of modern life, the reciprocal influence of individuals upon communities and of communities upon individuals render it necessary in a work of this kind to consider problems in economics, engineering, manufacturing, architecture, pedagogics, commercial intercourse, taxation, municipal government, etc. These have been brought together and studied in their bearing upon the subject and on each other, and are placed before the reader in as concise form as would seem possible. The bacteriology of the infectious diseases, the distribution of animal parasites, and the whole subject of the spread of disease by insects and other agencies form very important portions of the book. The care of the sick-room is discussed from the physician's point of view as a worthy department of therapeutics. The recent theories of immunity, artificial defences against disease, hygiene in cities, disposal of waste, disposal of the dead, hygiene of women at special periods, care of patient, and special nursing are headings which indicate some of the contents; but in a limited review of this many-sided treatise it is impossible to dwell upon the subject-matter in any details. Suffice it to say that we believe this to be one of those books which belong in the library of every physician.

In Volume VI we have a treatise on Foods, and as this subject has been dealt with by numerous authors, this addition to the list demands less notice from the reviewer than does the preceding volume of the series. The consideration of diet is not merely a question of how to feed the sick, but also of what is proper feeding for those who are in health; and this prophylactic phase of the subject is not forgotten by the author. In a food the chemical composition is important, but it does not indicate the suitability or unsuitability of that food for human consumption. The author, therefore, after describing the chemical nature of the fundamental food-bodies and their transformation in digestion, proceeds to determine which of these enter into the composition of the various food-stuffs (eggs, cereals, meat, etc.), and by what means and to what extent food-stuffs are rendered wholesome. First the diet in health at different periods of life, infant feeding, the diet of brain-workers, and the ill effects of food are given detailed treatment; then follow chapters on the feeding of the sick in the different infectious diseases, diseases of the blood, kidneys, digestion, circulation, etc. A chapter is devoted to feeding in nervous diseases, and includes diet-tables for neurasthenia and epilepsy. Undoubtedly the book is a mine of information about all the ordinary foods and their modifications, with their uses in health and disease, and even the subject of rectal feeding is not neglected. Each of the volumes in this series so far published is authoritative in its particular field, and the editor is to be congratulated on his choice of contributors.

W. A. Bastedo.

Medical Microscopy, designed for students in laboratory work and for practitioners, by T. E. **Oertel**, M.D., Professor of Histology, Pathology, Bacteriology and Clinical Microscopy, Medical Department, University of Georgia. With 131 illustrations. Philadelphia. P. Blakiston's Son & Co. 1902.

Microscopy is a new and growing science in which those who graduated more than a decade ago received for the most part but scanty instruction. But the more complete works on the subject offer a bewildering mass of material from which it is difficult for the unskilled to cull what will be of service to him. This has been kept in mind in the compilation of this treatise, and where possible the simpler processes have been chosen, and usually but one method given whereby to reach a certain result. The choice of material has been excellent, sufficient details being included to enable even a novice to fix, section and stain tissues, to prepare bacterial cultures or slides, to stain the blood or count its corpuscles, to determine the tubercle or other bacilli in the sputum, and to appreciate the significance of sediments in urine. The saliva, stomach contents, feces, vaginal secretions, etc., are not dwelt upon at length. The many illustrations, some of which are colored, are an important addition to the text; a few of the micro-photographs, however, would be unrecognizable without their labels. We can recommend this book as a practical guide not only to the uninstructed but also to those who have had their course in microscopical studies. W. A. Bastedo.—

# News and Notes.

THE STEVENS' TRIENNIAL PRIZE of Columbia University for original research was awarded to Doctors L. Pierce Clark and Thomas P. Prout of New York upon "Status Epilepticus: A Clinical and Pathological Study of Epilepsy."

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PATHOLOGIST AT CRAIG COLONY.—Dr. B. Onuf has been appointed resident pathologist at the Craig Colony for Epileptics at Sonyea, N. Y.

Dr. Onuf is a graduate of Zurich, Switzerland. He acted for two years as assistant to Prof. Forel at the Insane Hospital Burghoelzli at Zurich, afterwards devoting eight months to the study of ophthalmology with Prof. Haab in Zurich. Later he located in Brooklyn, N. Y., where he began to devote himself to neurology. He was affiliated with Dr. Sachs' clinic in New York for a number of years, and later with the nervous clinic at Long Island College, where he gave clinical and didactic instruction to the students. He served as neurologist to St. Catherine's Hospital and consulting neurologist of the dispensary of the Jewish Hospital in Brooklyn. From 1896 to 1902 he was associate in pathology at the Pathological Institute of the New York State Hospitals.

Among his scientific contributions may be mentioned a monograph written in conjunction with Dr. Joseph Collins, entitled "Experimental Researches on the Central Localization of the Sympathetic with a Critical Review of its Anatomy and Physiology."

In selecting a pathologist for the colony, the chief aim was to utilize the unusual opportunities given at the colony for studying the causes of epilepsy. The work is to be not only pathological but also clinical, and it is from this combination that fruitful results are hoped for.

RECENT PROGRESS IN MATTERS PERTAINING TO THE CARE OF THE INSANE IN THE STATE OF NEW YORK.—Some of the measures of improvement in the care of the insane of the State of New York carried through during the past few months by the Lunacy Commission are as follows:

1. The Pathological Institute has been reorganized and more than sixty of the medical men connected with the staffs of the fourteen State hospitals have been instructed during the past winter at the Institute on Ward's Island in the recent development of psychiatry along clinical, pathological, psychological and clinical lines. The legislative appropriation for the Institute is now $25,000 annually.

2. The hospitals have been opened to medical internes in the same manner as general hospitals. Last year sixteen clinical assistants entered the service in this way, and this year the number is nearer thirty.

3. The legislature recently passed the Lunacy Commission's bill for the appointment of a medical inspector to ensure a more thorough inspection of the thirty-nine institutions under its charge, viz.: 23 private retreats, 2 criminal asylums, and 14 State hospitals for the insane. Such inspection, especially of the private asylums, in which there are about 1,000 patients, has never been adequate.

4. To remedy overcrowding the Lunacy Commission proposes to construct a new hospital in the territory north of Albany and Troy on the colony system—a scheme similar to that of the Craig Colony for epileptics will be carried out. The site will be selected and plans made this year.

This colony for 1,500 to 2,000 patients should be ready inside of three years.

5. Three tuberculosis hospitals, each with a hundred beds, will be constructed this summer at a cost of $90,000 at Middletown, Utica and Binghamton, on the grounds of the State hospitals located there; and the plans made by Dr. Peterson and the State architect embody the main features of such hospitals described in the King Edward Prize Essays. In the meantime tent life for the tuberculous insane has been in vogue at the Manhattan State Hospital East (under Dr. Macdonald) for two or three years and for a shorter time at Binghamton and other of the State hospitals.

6. The country colony for a few of the working classes of the insane, as an offshoot of the Utica State Hospital, has been enlarged. A similar colony has been established at the Willard State Hospital, and two are in existence at the State Hospitals at Binghamton and Poughkeepsie.

7. A new departure this year is the creation of a summer camp for between 40 and 60 insane on the lake shore, about fifteen miles from the Rochester State Hospital, which is now in operation to the great delight of both patients and attendants.

8. The feature of nurses' homes having been found so useful at some of the hospitals two additional ones will be put up this summer, one at Kings Park State Hospital, Long Island, for 300 nurses, and one at the Gowanda State Hospital for 100.

9. Six or seven residences for superintendents and separate houses for the medical staffs will be put up this season at as many of the State hospitals, thus removing the officials from the central main buildings and utilizing the vacated space for patients.

10. A bill providing for emergency commitments, recommended by the Lunacy Commission, was passed by the legislature at the last session. Copies of this law have been sent to all examiners in lunacy of the State. It is believed that this will mean great good to the insane and prevent the all too frequent incarceration of urgent cases in jails and station houses.

11. The improved ration brought about during the past six or eight months, though entailing an additional cost to the State of a hundred thousand dollars per year or more, has added greatly to the comfort of the patients and to the satisfaction of the medical officials and various visiting boards.

12. A strong effort is being made by the Lunacy Commission to increase the number of deportations of alien insane, and through the efforts of New York State the federal government passed a law making the limit three years instead of one; i. e., an immigrant becoming insane within three years after landing in the United States may be returned to his own country.

13. The movement for the establishment of reception hospitals for acute curable cases in the large cities has gained strength. While the bill for the psychopathic hospital for New York city, which was to be the first of several such reception hospitals, failed to pass this year, it is believed that success will attend the Commission's efforts during the coming session.

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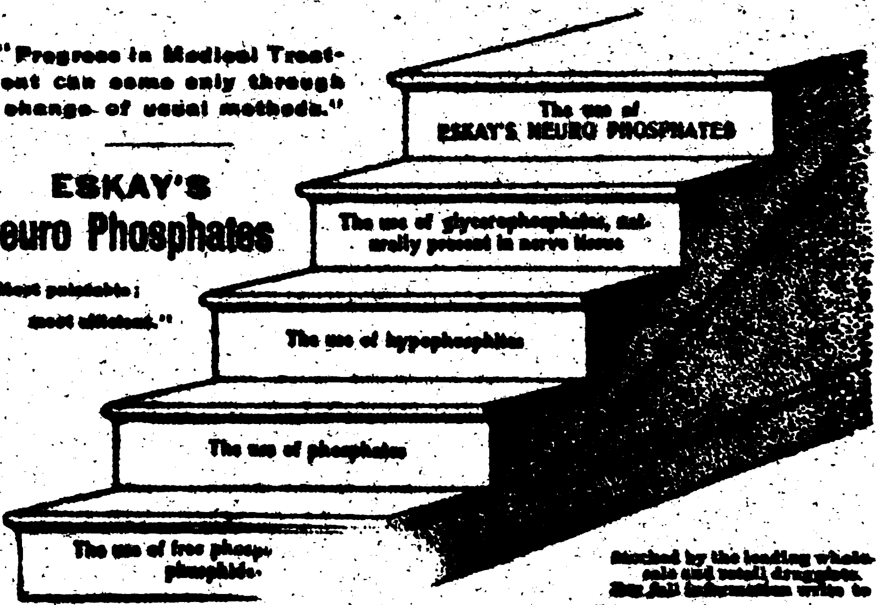
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Vol. 30 August, 1903 No. 8

# The Journal of Nervous and Mental Disease

The Official Organ of

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The New York Neurological Society
The Philadelphia Neurological Society and
The Chicago Neurological Society

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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

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Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

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ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, [illegible]

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THE
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1903

## Original Articles.

# MYOCLONUS MULTIPLEX AND THE MYOCLONIAS; REPORT OF CASES AND AN ATTEMPT AT CLASSIFICATION.

By Charles L. Dana, M.D.,

PROFESSOR OF NERVOUS DISEASES, CORNELL UNIVERSITY MEDICAL COLLEGE.

Myoclonia is a general term covering all the diseases in which myoclonus or muscle-twitching is a prominent symptom. This is the definition of Foster's Dictionary, and the use of the word in this general sense is the proper one. It has, however, been used by Seeligmüller (1887) to indicate the same thing as paramyoclonus multiplex, and Unverricht used it (1891) in about the same way.

Friedreich reported his case of paramyoclonus multiplex in *Virchow's Archivs*, Vol. 86, 1891. During the ten years following a number of cases which were more or less like his were put on record. When Unverricht published his "Myoclonia" (1891), he recognized that his cases were different from those of Friedreich, and he separated them into a group, called the "familial myoclonias." Following Unverricht, there came reports of a number of cases of myoclonia, some of which resembled this "familial" type, and some, more or less (rather less than more), the original case of Friedreich. The history of these is given in the monograph of Lundborg: "Ueber Degeneration und degenerierte Geschlechter in Schweden," published in 1891, and the more recent monograph of Clark and Prout, *American Journal* *of Insanity*, No. 2, 1902. The criticisms upon these various papers indicated different views. Thus Wollenberg and Boettiger concluded that the Friedreich type of case belonged to hysteria, and that there really was no such independent disorder as paramyoclonus. The "familial" type of Unverricht was thought to belong to the "degenerative chorea" of Huntington. In this view Moebius coincided. Schultz, on the other hand, placed the Friedreich type amongst the forms of convulsive tic and the Unverricht type amongst the degenerative choreas. Marie was of the opinion that paramyoclonus is a special disorder, and with this view Oppenheim, Bechterew and Strümpell appear to agree.

The view which I entertain differs somewhat from any of those expressed heretofore, though it is most in harmony with that of Schultz. It seems to me that the cases which were most in harmony with Friedreich's original case, belong to the group of myokymias, or neuro-muscular spasms without special locomotor effect, and are spasms dependent upon a disturbance of the peripheral motor neurones. There are certainly groups of cases of this kind, as is shown by those recently reported by Hunt[1]. Most of the other cases reported as paramyoclonus belong to the category of hysteria, or functional neuroses or to convulsive tics. The "familial group" of myoclonias described by Unverricht and Lundborg seem to me to belong certainly to the degenerative choreas, though they are pretty widely separated, as a rule, from the ordinary group of Huntington's chorea, being much more severe in their manifestations, and rapid in their course.

A further discussion of the reasons for this grouping will be given later.

The following cases illustrate four different groups or types of myoclonia.

1. Myoclonia of the spinal and peripheral type, including myokymia, fibrillary myoclonus of Kny, and Friedreich's paramyoclonus multiplex.
2. Myoclonia of functional and hysterical type.
3. Myoclonia of the convulsive tic type.
4. Myoclonia of degenerative chorea and epilepsy. (Myoclonus-epilepsy, myoclonia of family type.)

[1] Hunt: JOURNAL OF NERVOUS AND MENTAL DISEASE, June, 1903.

*Personal Cases.* Case I.—*Myokymia and myoclonus (paramyoclonus of Friedreich).* The following case showed very typically the phenomena of myokymia, with at times myoclonia and myotonic contractions with locomotor effect, irregularly distributed and involving muscles not under voluntary control. It is a case resembling in many ways that of Friedreich's, though the dominant feature was the myokymia, and resembles in this more the fibrillary myoclonus of Kny.

*Summary.*—Male, 23, workman, family and personal history negative. At 22 had twitchings of muscles, and painful spasms of legs and arms. Improved, but twitchings never entirely left him; cramps ceased recently. Entered hospital for general weakness, and diffuse pains in bones of legs, pelvis and head. No objective signs of any organic disease. Muscles of all parts of body in continual play of fascicular and fibrillary spasm, causing irregular tremor in fingers and toes, and sometimes locomotor effect of finger and leg. Fascicular spasm in thighs increases and culminates in myoclonus and myotonus of one or both hamstring muscles. Similar myoclonus in biceps. No painful spasm now.

Male, aged twenty-three years, single, Slav, was brought to Bellevue Hospital, complaining of pains in the legs and arms, and great general weakness. He stated that one year ago he had had a severe attack of pains which were called rheumatic. He at that time had also muscular contractions such as were still present and had had at times "cramp-like" contractions of the muscles of the legs and arms. He improved, but continued to have at times cramps, pain, and muscular twitchings. Of late this condition had grown worse. The patient was brought to the hospital, complaining of weakness and pains in the legs, thighs and head. He was a rather small man, somewhat emaciated, but with fairly good muscular development. His body was covered with a fine eruption which was found to be due to scabies.

As the patient lay in bed fine fibrillary spasms could be seen playing over nearly all the muscles of the body. They were very exquisitely marked in the face, where they affected especially the small muscles, such as the buccal, risorius, and nasal. The contractions were not very frequent, not more than one or two being seen at a time, and they occurred in single "flashes," jumping from one spot to another, they affected the two sides about equally. No general motor effect resulted. Similar phenomena occurred in the forearms and in the intrinsic muscles of the hands, the lumbricals and interossei. Here they recurred frequently, causing slight locomotor effect and a condition like a fine tremor, only the fingers were also irregularly flexed, abducted and adducted. When the long flexors were involved the fingers were rhythmically flexed a trifle, the movement sometimes running into a tonus with contraction of the fingers. The movements occurred in the forearms, arms, shoulders, pectoral, abdominal and posterior trunk muscles; here they were more violent, but were less frequent and caused no locomotor effect, except that occasionally the biceps would move the forearm. The calves and intrinsic muscles of the toes were affected just as were the upper extremities. On making the patient lie on his face the movements could be seen most strikingly of all in the muscles of the calf, posterior part of the thigh, and in the buttocks. Here then were frequent fibrillary contractions and also wave-like contractions running along the muscle from center to periphery. On flexing the leg at right angles to the thigh, the inner hamstring muscles would begin to contract more vigorously and finally ended in a distinct myoclonus or more often in a tonic contraction which held the leg flexed for several seconds. The same thing occurred in the outer hamstring muscles and sometimes in both, holding the leg in flexion. Thus there were here fibrillary contractions, wave-contractions, finally ending in a distinct clonic or tonic contraction of the whole muscle, sometimes with locomotor effect. The phenomenon occurred in muscles such as the inner hamstring, not under direct voluntary control. The contractions could be brought out by manipulating the leg, and by blows of the hammer. They were unaccompanied with pain, and the flickering spasms occurred at times 40 to 60 times a minute. The myoclonus and myotonus were observed only when the leg was flexed and the muscles relaxed. A similar condition but much less marked was present in the biceps. The myokymia continued during sleep. There was no atrophy, contracture or paralysis. The general muscular irritability was much increased and immense myoid tremors could everywhere be easily produced. The electric reactions were not tested. The muscular power was fairly good. The deep and skin reflexes were normal. There was no sensory disturbance of any kind. The special senses were normal. He had no stigmata of hysteria. The patient complained of general pains, which seemed to be mainly in the bones. There was no tenderness or swelling of joints or muscles. His mind at first seemed at times confused. He presented no objective symptoms of any visual disorder, or of disease of the central nervous system. The blood and urine were normal, eosinophiles normal. He could walk, but his weakness kept him in bed. He improved and the eruption grew less under mercurial treatment, but the muscular phenomena were unchanged.

Taking the whole history of the case it will be seen that he had painful cramps as in Friedreich's case, clonic twitchings of the muscles of the thighs and arms, not of the peripheral segments, muscular contractions of the whole body of a muscle, sometimes with and sometimes without locomotor effect, and that muscles were affected not under voluntary control. In all these respects, the phenomena were like those of Friedreich's case, but in addition he had a widespread and remarkable myokymia, involving nearly all the muscles. The spasms could not have been of cerebral origin, certainly not cortical, and it seems to me that the seat of the trouble must be either in the spinal cord or peripheral motor neurone,—which is the pathology attributed by Friedreich to his case.

It shows a close relation between ordinary myokymia and the severer types of bundle clonus, to which the term paramyoclonus multiplex, if used at all, should be applied.

Case 2.—*Paroxysmal myoclonus of functional (hysterical and family type. Summary.*—Male, aged forty-two; mother suffered from similar attacks. Healthy till the age of eleven years, when he was kicked in the head by a horse. One month later seizures began, which have lasted thirty-one years, gradually lessening in severity. Seizures come on in groups of short attacks lasting one to five days. Seizures are characterized by some fibrillary tremor, irregular clonic movements, tonic contractions with and without locomotor effect and violent rhythmical movements affecting the face, neck, shoulder and arm muscles and, to some extent, the legs. Muscles of the back are sometimes involved. The muscles involved are symmetrical but not synchronous in their clonic movements.

E. P. B., single, farmer, aged forty-two. Father healthy but of nervous temperament; no phthisis or inebriety, no nervous or mental disease in family. His mother was a healthy woman but during her pregnancy she had attacks of muscular twitchings and jerkings known as "shaking spells." These came on periodically during the day and even interfered with her sleep. They occurred in the latter part of the pregnancy during which she was carrying the patient. After the birth of the child the attacks disappeared. The patient had one healthy sister and lost one sister when she was a baby. He was perfectly healthy until his eleventh year. He was then kicked on the left side of the face by a horse and knocked down, and remained unconscious for three-quarters of an hour. About one month later while sitting quietly in the house he had the first attack of his spasmodic trouble. These attacks would last with intermissions for twenty-four hours, beginning in the night sometimes and lasting till the next night. He would then go for a few days or a week without any, then they would come on again. At times he would have them with short intermissions of a day or two, all one summer, then they would go away for some weeks, then return again. His trouble continued in this way with various remissions till first seen by me, when he was thirty-four years old, at which time he had had the attacks for twenty-three years. During all this time he had no epileptic attacks and nothing resembling epilepsy. He had managed to get his education and attend to his work as a dairyman, spending some time occasionally at sanitariums for treatment.

The year before I saw him he had had an attack of sciatica affecting both legs. This lasted for a month. He had a second attack about four months before I saw him which had lasted all summer. On account of the pains in the legs and the other attacks, he had lost weight, being reduced from 158 to 105 pounds.

When first seen by me at my office he was not having the attacks, and presented the appearance of a nervous, rather anemic and badly nourished man. There was no objective trouble with the muscular system, though the muscles were rather poorly developed. There was some tremor of the hands, rather more in the right, and a little tremor in the legs. The dynamometer showed 30 for right and left hands. There was some fibrillary twitching of the muscles of the back. The skin reflexes were exaggerated. The deep reflexes were normal. There was no disorder in the sensory sphere, the special senses were normal. He had no anesthesia nor ataxia. Bladder and sexual organs were normal.

When the attack came on he was seized with certain rhythmical movements of the head; the hands would shake rhythmically, also the feet, and the lids would quiver. The muscles of the trunk were not involved, but the muscles of the back of the neck would contract and harden, pulling the head back slightly in clonic twitches. When called to see him in one attack at night I found his head moving rapidly antero-posteriorly. Also his arms would be shaken to and fro, but not synchronously, one arm would move more than the other. The trunk and leg muscles did not at that time move very much, though his eyes would blink and his feet and legs would be twitched in irregular clonic movements. He had no pain. The spells would last half a minute, then recur every minute or two, and in this way they would go on for one to five days. They would keep him from sleeping, unless he took some narcotic, then he would go to sleep and the attack would cease. During an attack there was considerable palpitation of the heart, he perspired profusely after the attacks and they would leave him exhausted. Consciousness was never disturbed.

The muscular movements of these attacks, therefore, consisted of violent tremors, tonic contractions of the body muscles without motor effect, clonic contractions irregular in distribution and fibrillary contractions of the body of the muscle which could be observed in between the attacks as well as in the most violent, to precede them. The attacks would occur in short intermissions, every half minute or so, and would last several minutes, and this would go on for from one to five days. These groups of attacks would thus recur every few days, sometimes every week or two, and sometimes intermit for several months.

The patient was not improved by the various measures employed by me, including hyoscine, hypnotics and tonics. He was hypnotized five times, without any special benefit. He was under my observation for one month. He then disappeared, and I heard nothing from him till a short time ago, five years after my first observation, when he wrote me that his general health was excellent and the nervous trouble very much milder, so that when the attacks came on he practically suffered little He states that when they come on he goes to bed, as he is more comfortable there, and passes the time reading until they are over. He had had no epilepsy or hysteria. He is satisfied that it is a family disorder and accepts his affliction philosophically.

Case 3.—*Myoclonia of convulsive tic type. Summary.*—Male, aged forty, no heredity. Abscess of thyroid following typhoid fever; soon after developed clonic, tonic and fibrillary spasms, mainly clonic, affecting first and mostly muscles of abdomen, back and neck; less, those of face, and of forearm; least, those of lower extremities; cease during sleep; no pain; spasms are mostly coördinate, causing grimaces, wry-neck, movements of shoulders and trunk, isolated muscle spasm without locomotor effect, fibrillary and fascicular spasm not marked. Is able to walk and use his hands in delicate work. Spasms vary in intensity without absolute cessation. In four years has gradually improved. No organic disease, general health fair; no epilepsy, mental condition good.

R. M., aged forty years, traveling salesman[1]. The family history is negative and there is in particular no evidence of disease of the nervous or muscular system in the ancestry. Except for the usual diseases of childhood the patient has been well all his life. He denies venereal infections and has been a man of moderate habits. In November, 1896, he had typhoid fever, which kept him in bed for six weeks. The disease ran a mild course without serious complications. During its course, however, a swelling over the thyroid region began to form, which became an abscess. Eight weeks afterwards this abscess broke spontaneously, discharging pus. At the same time the right lower extremity became "awkward and lame," and the patient began to stoop. Shortly afterward he began to have what he called "spasms in the stomach." He was treated with hyoscyamin for this. Slowly the spasmodic discharges began to spread, and by March, 1897, they were very general. In May, 1897, he was treated with thyroid extract, and he says that he was delirious and out of his mind for four weeks. In August, 1897, another tumor of the size of an egg began to form in the thyroid gland, in the location of the previous one. This tumefaction was removed by Dr. Lilienthal, on October 5, 1897. According to a report received from the Mt. Sinai Hospital this tumor was a thyroid cyst. After leaving the hospital the patient says he was well and free from spasms for a period of ten days. They then returned and became continuous, so that he was admitted in August, 1898, to the Montefiore Home, having been ill for over one and a half years. During this time, the spasms, which began in the arms and abdominal muscles, had become generalized. The muscles of the tongue, face, throat and neck had become involved. He would thrust out his tongue and grimace, as in tic, or would throw his head back or twist it to one side, as in wry-neck. He had no pain at any time. Immediately after getting up in the morning the spasms were comparatively slight, but they increased in intensity and reached the climax about four o'clock in the afternoon. Upon going to bed the patient was compelled to take a hypodermic injection of gr. 1-100 hyoscine, and then had a good night's rest. Still at no time of the day, except when he was asleep, did the spasms cease entirely. They were worse when he was excited, and were mildest when he was quietly occupied. He was able to shave himself and to do various kinds of dexterous work with his hands.

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[1] Notes are by Dr. Joseph Fraenkel and myself.

*Status praesens.*—Patient is of medium size, rather poorly nourished, and his skin shows various tattooed figures. He is otherwise free from scars, except a small one over the thyroid region, slightly adherent to the deeper tissues. The skin is of a dirty hue and not very elastic. The muscles are fairly well developed; there are no localized or individual hypertrophies apparent; the muscles are, on the contrary, when not agitated by the spasms, rather flabby and hypotonic. The usual attitude of the patient is a bending forward one, amounting at times almost to a complete doubling up of the body. The head is bent forward to the left and the chin turned to the right. When walking the attitude is the same. He seems to be drawn to the left and shows a sort of left-sided latero-pulsion. It is very difficult for him to assume the erect posture on account of spasms of the abdominal muscles, and attempts increase the spasms.

Temperature 99½ (by mouth); pulse between 130 and 150; pulse is soft, volume rather small; respiration varies from 12 to 18, the rhythm and vigor being constantly interfered with by the spasms.

The thoracic and abdominal viscera are normal.

When the patient is observed, while sitting quietly on the chair, there is a ceaseless and varying play of muscular spasms. These spasms both affect individual muscles and physiologically connected muscle groups, and are essentially clonic, running often into a tonus. The muscles of the trunk, neck and face are affected most. The contractions of the extremities are very much less than those of the rest of the body and the upper extremities show more violent and frequent spasms than the lower; in fact, the lower extremities are, but for the occasional clonic spasms in the quadriceps, almost entirely free; in the upper extremities the muscles nearest the shoulder joint are affected most. The muscles of deglutition and mastication are also affected. There is frequent grinding of the teeth, and swallowing is sometimes interfered with, morsels dropping out of the mouth and fluid passing through the nostrils. There are at times noisy and irregular respirations, due to spasms of the diaphragm. After a number of these severe spasms the patient is covered with perspiration and is considerably exhausted. The frequency of the spasms varies from 10 to 60 per minute.

In the psychical sphere it is noteworthy that the patient cries easily; otherwise no defects are apparent.

Cranial nerves, special senses, gross motor power and cutaneous sensibility are absolutely undisturbed.

Pupillary and skin reflexes are good; tendon reflexes of moderate intensity and easily demonstrative when free from spasmodic contraction. Rectal and vesical sphincters are normal; the patient states, however, that he is unable to urinate except when he is sitting down, and that the spasms cease entirely during defecation and micturition.

The sexual functions are normal. The electrical excitability of muscles and nerves is rather increased, the direct stimulation of the muscles is easily exhaustible. The mechanical irritability of muscles is slightly increased.

A *second examination* was made three months later. There is now a persistent tonic spasm of the abdominal muscles which holds the patient in a stooping position. The recti muscles are thus contracted nearly all the time. They relax only during sleep and for short periods during the day. The pectoral muscles show frequent, bilateral clonic twitchings, at times at the rate of about 50 a minute. Choreic, irregular, not bilateral twitchings are seen from time to time in the muscles of the arms; there is here and there a twitching of the biceps and triceps muscles alone, particularly on the left side. The platysma myoides shows clonic twitchings on the right or left side, but not simultaneously. The right sterno-cleido-mastoid muscle and upper trapezius undergo clonic twitchings and tonic spasm, pulling the head to the left, as in wry-neck. Similar spasms of the left sterno-cleido-mastoid muscle frequently occur, turning the head to the right.

On the whole, the left spinal accessory nerve is more affected than the right, and the spasms are more tonic on this side. Clonic tonic bilateral facial spasms producing prolonged (i.e., for two seconds) grimace occur, and along with them there occur irregular clonic contractions involving the tongue and face muscles.

The patient used to grind the teeth when asleep, but now there is a continual grinding of the teeth and smacking of the lips, as in forms of tic and chorea. There is no nystagmus or disturbance of ocular muscles.

The gluteal muscles go from a state of wavy, fibrillary twitching into a tonic spasm when the patient is put on his back (See Case 1), while the irregular choreic contractions of the lower extremities occur at the same time. To mechanical irritation most of the muscles respond with myoid tremor.

*Third examination,* June, 1900, three and one-half years after the onset of the disease:

The patient's condition is somewhat improved. Still he is in almost constant movement. The spasms continue to affect chiefly the musculature of the neck, shoulders and upper arms, and trunk, the lower extremities being comparatively free; the deep muscles of the back are not affected as much as at the last examination. The number of convulsions per minute is variable, the finer fibrillary contractions occurring as high as forty; the coarser grosser movements occur as high as ten per minute.

The spasms are mostly clonic in character, but also fibrillary and tonic. These movements result in the flexion and extension of the forearms, abduction and elevation of the arms, a flexion of head on thorax, and very slight flexion of trunk on thighs. The peripheral portions of all four extremities are least affected, the movements being greatest in the upper thoracic portions of the body and neck. The diaphragm is still irregularly affected, causing irregular breathing, and to some extent the speech, patient breathing and speaking in intervals. The movements are to some extent symmetrical as to muscles involved, yet not as to time of involvement. Contractions occur irregularly, affect the biceps, then jump to fingers, then to head, then to forearms. They affect usually coördinate groups of muscles, but also individual muscles. Fibrillary twitchings, involving very small limited portions of muscle, and twitching of distinct muscle bundles are seen, the rest of the muscle being apparently at rest. The movements of patient are well nigh continuous under observation, though when unobserved distinct intervals of quiet are apparent, though even then fibrillary contractions are evident.

Effect of position upon movements.—Recumbent posture diminishes movements of trunk, but to a less extent other movements.

Standing increases markedly movements of trunk and somewhat the movements of legs.

Noteworthy that muscles most concerned in standing are least affected, but upper extremities are threshed wildly about.

Standing on one foot possible for a length of time, equal to that possible to a normal person.

Standing with eyes closed, feet together is normal.

Fixation of attention steadies the spasm.

Volitional movements steady spasm, patient being able to shave himself and to do delicate manual work. Things do not drop from hands.

Irritation of skin.—Tickling, pinching, sticking, pressure and cold increase the twitching.

Emotion has a decided staying influence, he asserts.

Alcohol has no effect. Sleep inhibits the movements entirely. Spasms never rhythmical. Tonic spasms are apparent in anterior muscles of neck, yet upon closer investigation they consist of many rapidly occurring clonic contractions.

Sensibility.—No paresthesias. Pains.—In right side of neck and lower and upper extremities. Tactile sense, normal. Pupils equal and normal. Ocular movements normal, but affected by general spasms. Facial innervation equal. Pharyngeal reflexes diminished. No reflexes in upper extremities. Musculature fair in volume, no atrophies. Myotatic irritability increased. Myoidism excessive.

Abdominal reflexes lively. Cremasteric present. Knee-jerks markedly diminished. Achilles ditto. Plantar reflex present;

flexor response. Gross motility normal. No ataxia. Electrical examination: normal reaction.

Pulse 108, full. Respiration variable, 22. Marked prominence of superficial veins of upper extremities and abdomen (caput medusæ). Lungs normal. Heart: no murmurs. Liver apparently enlarged.

*Fourth examination* on March 25, 1902. The patient walks well, but his position is that of extreme kyphosis. There is continual nodding spasm of the head with occasional grimaces of the face. There are no spasms of the eye muscles. The neck muscles are occasionally contracted in tonic spasms which relax at the end of three or four seconds. There are continual rhythmical tonic spasms of the pectoral muscles, drawing the shoulders together, and of the abdominal muscles, pulling the trunk forward. The upper arms are affected in the same way, with occasional tonic spasms involving the whole musculature of the arm, so that there is no actual movement of the forearm. The forearms and hands are not affected by any spasms. There are movements of the back muscles drawing the scapulæ together. He has occasional movements of the intercostals and diaphragm, and owing to this he has, at times, difficulty in breathing. His spasmodic condition, has, on the whole, improved, but he has lost flesh and looks anemic and weak.

At the present time, 1903, seven years since the malady developed, he is much improved, but has still facial grimaces, and neck spasms like those of wry-neck. Mental condition good.

Case 4.—*Myoclonia of type of degenerative chorea, or myoclonus-epilepsy. Summary.*—In 1890, I reported in *Brain*, p. 71, a case of "Chronic Chorea with Autopsy." The patient, a male, aged eighteen years, negative family history, began to suffer with attacks of the chorea of Sydenham at the age of six years. These became chronic and associated later with epileptic attacks. For more than six years the patient suffered from attacks of chorea of a mild type, and occasional nocturnal epilepsy, which did not prevent him from going to school and afterwards to work. The movements increased in violence and extent, and he was finally, at eighteen years, obliged to give up work and go to a hospital. At that time he had not only clonic and choreic movements, but tonic contractions of the muscles of the trunk, fibrillary and wave contractions of the muscles, tremor and not infrequently attacks of an epileptic character. He developed pneumonia and died. I reported the case as one of chronic chorea with epilepsy, but as it appeared in the later stages it would certainly be classed amongst the cases of myoclonus epilepsy, if that classification is adopted.

Case 5.—*Myoclonia of type of degenerative chorea. Summary.*—In 1894, under the title of "The Microbic Origin of Chorea, Case with Autopsy," *American Journal of the Medical Sciences,* January, 1894, I reported a case of a very singular character in a man aged thirty-four years, who had a good family history, who developed rheumatism in the tenth year followed by Sydenham's chorea. This chorea recurred every two or three years, until finally, eight months before he was seen by me, he developed violent choreic and clonic movements, affecting all the muscles of the body, with tonic spasms of the neck and back at times, and rhythmical movements of the head and arms. The case in these last stages resembled Case 2. He subsequently died and an autopsy was made. This man did not have any epileptic attacks, but had a combination of choreic convulsive tic, tonic and rhythmical spasms. The multiple myoclonus, however, was in the last year of his life the dominant feature of the case.

Cases 6, 7, 8.—*Cases of myoclonia of the type of convulsive tic.* In Thompson's "American System of Practical Medicine," under the head of "Choreic Tic" I reported a series of three cases of children who began with what appeared to be ordinary choreic symptoms. In these cases there was first a condition of ordinary choreic movements affecting the face and arms, later tonic and choreic spasmodic movements of the muscles of the neck, causing the patient to appear to have a combination of convulsive tic, or wry-neck, and chorea.

These three cases I called forms of choreiform tic, because beginning with a chorea, like that of the infectious or Sydenham's type, they later developed symptoms of wry-neck and had other tic-like spasms. They belong with the myoclonias of the convulsive tic type, like Case 3.

I have tried to determine the features of myoclonic disorders which ought to be brought out in a description and utilized in classification. Perhaps I have not all, but they seem to be about as follows:

1. The cause.
2. The association with other diseases.
3. The mode of onset.

4. The duration.

5. The persistence or remittance, or intermittance.

6. The groups of muscles or the limbs affected.

7. The symmetry, or bilateral synchronicity.

8. The quickness, or electric character of the spasm.

9. The involvement of muscular groups used coördinately.

10. The involvement of incoördinated groups.

11. The involvement of single muscles not under voluntary control.

12. The motor, or locomotor effect of the twitch.

13. The fibrillary twitching and the twitching of bundles or large masses of the muscle.

14. The predominance of 10, 11, 12 and 13, if several types exist together.

15. The association with tonic or rhythmical spasms, or with epilepsy and mental deterioration.

16. The association with pain, atrophy or paralysis.

17. The condition in sleep, or under anesthesia.

18. The control of the spasm by the will.

19. The influence of emotional excitement on the spasm.

20. The termination.

It is assumed that there is no organic disease and the trouble is not symptomatic of such condition.

Now applying all the tests of character to known and common spasmodic disorders, we find an agreement in the peculiarities of chorea, as it ordinarily develops, i.e., the infectious or Sydenham's type. Nor is there difficulty with symptomatic or degenerative chorea of Huntington.

I think that French and American neurologists also agree pretty well as to what is meant by the different types of spasmodic tic, tic *coordiné*, or the tic neurosis of Collins. It includes, to my mind, not only facial tic, but torticollis, and many local tics, as of the diaphragm and larynx, of the trunk muscles, and it may even be generalized as in what I have called progressive or "choreic tic" (myoclonia of the tic type). It is degenerative in origin, and it is a cortical disorder. (I admit cornual or nuclear tics.) It is often a kind of occupation or habit neurosis. It is slow in onset, chronic in character, very nearly continuous in its manifestations. It usually affects the face, neck, the larynx, articulatory muscles, diaphragm or larynx, but it may affect the legs. (I have reported such a case in a boy who had a jumping tic, as well as a speech and facial one.) It is not symmetrical in its distribution. The movements are lightning-like and cause locomotor effects.

There are with it usually no fibrillary twitchings or bundle movements. The clonus may, however, in severe cases, be prolonged into a tonus, and this is the tendency in severe cases Rhythmical tremor and epilepsy may be associated with it. There is no pain, atrophy or paralysis, but rather a tendency to muscular hypertrophy. It ceases during sleep, it can be controlled in a measure by the will, and is made worse by excitement. It is only rarely curable.

The majority of reported cases of paramyoclonus multiplex can be placed in this group as myoclonias of the tic type.

There is another group of myoclonic disorders which has passed under the name of paramyoclonus multiplex.

Its characteristics, if there is such a disease, are claimed to be these:

It is caused oftenest by fright or some psychical disturbance.

Its onset is rather sudden and the duration rather chronic, i.e., months or several years. The spasms intermit, or remit. The muscle groups affected are mainly those of the shoulders, upper arms, thighs and trunk, and the face and periphery are rarely involved. Both sides of the body are affected, but not synchronously.

The movements are lightning-like, and the muscles affected are those of physiological groups, but single muscles and sometimes those not under control of the will may be affected. There is usually decided locomotor effect. There may be tonic and rhythmical spasm (Cases 4 and 5 or group I of Unverricht). The movements cease during sleep and are to a certain extent controlled by the will and increased by emotional excitement.

This type of disorder is distinguished from the tic neuroses by the intermittent duration of the spasms, the involvement of large groups of muscles producing bodily movements and tremors, the absence of spasm of small muscle-groups, causing unilateral facial grimaces and wry-neck movements. The rather favorable course. The neurosis should be placed among the hysterical, or functional groups. My case (Case 2) shows it *may* be a family type and not associated with hysteria.

A fourth group of myoclonias includes myokymia and allied spasms. It is characterized by persistent fibrillary and wave-like contractions of the body of the muscles. Higher and more intense types of this condition show also clonus and tonus of individual muscles. The so-called Friedreich's myoclonus multiplex, the fibrillary myoclonus of Kny would go in this group. They are essentially intrinsic spasms, grosser kinds of fibrillary tremor. So far as I can make out Friedreich's original case was one of this kind, and I agree with Dr. Hunt that they are peripheral or spinal in origin.

Thus I would group the myoclonias under the head of:

1. Myoclonia of Friedreich, or peripheral type, including myokymia.
2. Myoclonia of the functional or hysterical type.
3. Myoclonia of the convulsive tic type, this last type being characterized by many associated spasms, choreic and tonic.
4. Myoclonia of the degenerative chorea or "familial" or myoclonus-epilepsy type, which is closely related with the third.
5. Myoclonia, of the type of the infectious and symptomatic choreas. Although there is sometimes a mixture or fusing of the above groups, they are essentially different disorders, and as a rule can all be easily recognized and easily distinguished from each other. All cases of paramyoclonus multiplex, reported as such, can be grouped in one or the other. Hence there is no need of that much misused term.

*The Myoclonias and their Synonyms.*—The following somewhat interesting list of myoclonias and choreas shows how extensive the nomenclature of muscular spasm has become. I have tried to assign them to the five groups above enumerated. Probably the arrangement is not perfectly correct, or complete, but it is approximately so. I have excluded rhythmical spasms because myoclonia means essentially an irregular spasmodic disorder, a muscle "tumult" (*klonos*), or twitch, or succession of twitches, not an orderly or rhythmical phenomenon like a tremor, though rhythmical spasm may be part of the general picture.

I. Paramyoclonus multiplex, of Friedreich; astasic myoclonia, of Vanlair; multiple spinal myoclonus, of Lowenfeld; fibrillary chorea, of Morvan; fibrillary myoclonus, of Kny.

II. Functional, or hysterical myoclonus multiplex, chorea major, chorea electrica, of Henoch.

III. Myospasmia; memory-spasms, of Friedreich; habit chorea; chorea variable des degenerès; convulsive, or spasmodic tic; tic general; Tourette's disease, myriadrit; palmus; tic neurosis, of Collins.

IV. Degenerative chorea, hereditary chorea, Huntington's chorea; myoclonus-epilepsy; myoclonus, of Unverricht, of "familial" type; myoclonia congenita of Seeligmüller (?); hereditary degenerative chorea of Sachs.

V. Infectious chorea, chorea minor, Sydenham's chorea; chorea electrica of Dubini (?); chorea electrica of Bergeron; senile chorea of Gowers (?).

The stasic myoclonia of Vanlair is apparently a myoid tumor.

As there has been so much discussion over the nature of Friedreich's original case, I append here a translation of the history without adding the author's comments.

## APPENDIX.

### TRANSLATION OF THE ORIGINAL RFPORT OF A CASE OF PARAMYOCLONUS MULTIPLEX. BY PROF. FRIEDREICH.

Ludwig Berlein, fifty years old, carpenter from Mosback (Baden) was taken ill in 1877, with sudden fever, and the appearance of a right-sided pneumonia, following which were developed a chronic induration with shrinking of the upper lobe of the lung, and a dilation of the bronchi of the same lobe. From that time on the patient suffered from dyspnea, chronic cough and mucopurulent expectoration. Lately, also, there appeared progressive emaciation and weakness, which interfered with his work, so that the patient was admitted to the clinic on January 17, 1878. Under the use of liver oil, morphine in the morning, strengthening diet and careful nursing the patient's strength and nourishment improved. Cough and expectoration diminished to a slight remainder, and the patient was able to return to his previous occupation on the 8th of April. But in the fall of the very same year the above-named phenomena returned with renewed violence, so that after a temporary stay in a few other hospitals the patient was for a second time admitted to the clinic on April 29, 1880. The symptoms of bronchitis in the region of the cirrhotic parts of the lung were developed to a high grade, while the lung itself did not expand any.

The left lung was perfectly sound. No fever. No change in the heart or abdominal organs. Two of the sick man's sisters were said to have succumbed to pulmonary tuberculosis.

During the first as well as during the second stay of the patient in the clinic a most peculiar affection of a set of muscles in the upper and lower extremities became noticeable. This showed itself in the form of short rapid contractions, reappearing at short intervals, which affected the biceps and triceps of both arms, and supinator longus of the forearms exclusively.

At the same time chronic spasms showed themselves in the upper part of the thigh involving the vastus externus and internus and especially the rectus femoris, also to a less extent the abductors, and further the biceps and semitendinosus were affected, while the rest of the muscles of the upper part of the thigh were entirely intact. Here and there a muscle of the back or face was slightly affected by this disturbance, which confined itself to that particular muscle exclusively.

Here it was not merely fibrillary or fascicular twitchings but also spasms which involved the whole of the muscle which swelled forward in a mass, and bulged every time, even when the contractions of the muscles were not marked enough to produce a noticeable locomotor effect on the limbs to be moved. Only at times, at an unusually marked contraction, one could notice a slight change in the part to be moved, e. g., during many contractions of the biceps a slight flexion of the forearm or during some of the spasms of the supinator longus a slight supination of the hand.

The spasms of the rectus femoris produced a short and rapid forward movement of the knee joint, that of the biceps and semitendinosus a rapid movement of the part affected, following the tendons which limit the popliteal space.

The spasms, however, were unrhythmical throughout and varying in extent, even though they were separated from each other only by short intervals. (It may be mentioned here, by the way, that by placing the stethoscope on the affected muscles in the moment of their contraction a loud and clear sound, resembling the first heart sound in every respect, could be appreciated.)

Although the affection offered a perfect symmetry in its art, as the very same muscles were affected on both sides, yet every muscle showed a complete independence in relation to its individual contractions. Now it was this muscle, now that muscle, which twitched all for itself without any relation to the others, and if it sometimes happened that the contraction of a muscle on one side took place synchronously with another muscle of the same or other side, or even at times if two symmetrical muscles contracted at the same time, it was only an accidental coincidence, as it may be understood that such an occurrence was made possible by the frequency with which the spasmodic contractions appeared in each single muscle.

The frequency and intensity with which the contractions of each single muscle ensued were not the same on different days or at different times of the day, and in this regard a considerable difference could be noticed. During the times of greater agitation there were 40 to 50 contractions per minute in one and the same muscle, while during the hours of greater rest only 10 to 20 contractions could be counted. Only rarely did it happen that the spasm ceased entirely, or almost entirely, within one-quarter or one-half hour.

Were the spasms of less frequency, so were they also of less intensity, and vice versa. Frequency and intensity always stood in equal relation. It often happened that in one or the other muscle, contractions followed each other very quickly with increasing rapidity, and passed over into a short tetanus lasting about one or two seconds, which was always accompanied by painful sensations in the muscles. In general, the spasms were strongest and most frequent when the patient lay quietly in bed without carrying out any active motions of the extremities. Especially during the evening when the patient attempted to give himself over to the quiet rest of slumber, these cramps in the contracting muscles increased until they produced painful sensations, and thus going to sleep was rendered difficult.

However, if sleep once set in, all the spasms ceased entirely while it lasted. Yet it often happened that the patient was frightened out of his sleep by a sudden very painful, crampy movement of both legs, so that the latter were jerked up against the abdomen. The adjoining individual contractions of the above-named muscles were then always of singular intensity, and it lasted hours until these quieted themselves. But many times, even during the day, although much more rarely than during the night, when the patient lay quietly in bed, there appeared flexion cramps of this sort in the lower extremities, similar to those that are known to appear in cases of acute myelitis.

As soon as one undertook real motions and prolonged active contractions of the affected muscles the spasms immediately disappeared, and the former movements were in no way injured by them.

Thus the patient was not at all disturbed at his work; as soon as the muscles weakened and began to rest, the spasms returned. So during the active extension or flexion of the forearm there were no traces of any spasms in the biceps and triceps of the same side, while the spasm of these muscles of the other quietly lying down continued without weakening.

The lower extremities were in analogous condition, only that in the thigh the painful contractions of the affected muscles continued also during upright position, but here also they entirely disappeared during locomotor actions, as walking. The rough motor power of the affected muscle was in no way affected, and only with the greatest effort could one flex an extended or extend a flexed extremity of the patient against the patient's will.

Station and locomotor coördination were very little affected.

The electric irritability of the affected muscles, as well as those innervated by the same nerve trunk, according to repeated examinations by Prof. Schultze with both kinds of currents, was normal throughout; the mechanical irritability also showed no deviation, and a stroke on the biceps, etc., either by a percussor or with the edge of the hand, showed an idiomuscular swelling in no way higher or more prolonged than one often sees in most healthy individuals. On the other hand, there was an unmistakable increase of reflex irritability of the muscle to skin irritation. Even the effect of cool air on the skin of the exposed extremity increased the frequency and intensity of the spasm. On one occasion during a prolonged exposure, while the patient was being examined, the whole right arm was in painful and violent convulsive tremor, which lasted for one minute. The other skin irritations, such as pinching, pricking, etc., also increased the spasms, and as one would grasp the arm with his hand and squeeze firmly for some time, the spasms of the biceps and triceps would immediately become stronger and more frequent, and often followed each other in such rapid succession that they passed over into a short, lasting tetanus. Here, as it was mentioned above, there could be found no ground for the existence of an increase in the direct mechanical irritability of the muscles, and so this phenomenon was evidently the expression of an increased reflex irritability of the muscles. From the plantar surface one could also bring forth unusually strong reflexes of the lower extremities, and even a slight tickling was sufficient to call forth convulsive tremors of the legs, which repeated themselves after coming to rest a number of times. Tickling of the inner side of the thigh showed marked, although not unusually strong, cremasteric reflexes.

As far as the tendon reflexes were concerned, striking over the tendo-Achillis call forth only very weak contractions of the muscles of the thigh, and no foot phenomena could be brought out, so that in this respect the normal relation existed.

In strong contrast to this there was a marked increase of the patellar reflex; even a slight tap on the tendon of the quadriceps-extensor or ligamentum patellæ was sufficient to produce the most lively and many times repeating extensions of the legs; and when one stretched the tendon of the quadriceps by pulling the patella down, the spasm of the affected muscle appeared, and there ensued painful cramps, which of themselves passed over into an intense tonus, during which time the extremity was strongly extended and the patella drawn forcibly upwards.

On the other hand, irritation and extension of the ligamentum patellæ by pressing it from below upward over the knee joint had no effect, nor were the spasms of quadriceps influenced by it in any way.

All these phenomena which have been described, as well as the muscular spasms and increased skin and patella reflex, were more marked on the right than on the left side. There were no symptoms which would refer to any vasomotor or secretory disturbances. The skin sensations remained normal in every way, and in the same way touch and pain, and differences in temperature were appreciated and localized quickly and precisely. Few phenomena appeared which could be interpreted to mean an injury to the deep muscular sense. There was no atrophy of the affected muscles, excepting the general emaciation and bad nourishment of the whole body, referable to the chronic lung disease.

The physical functions were normal; headache, vertigo, etc., bothered him most. The activity of the higher mental senses was also undisturbed. The pupils were of equal size, somewhat narrow, their reaction to light and accommodation active and normal throughout. There was no fever at any time. Pulse 80 to 90. Some complaints of palpitation. The digestive functions were undisturbed. The secretion and excretion of urine were normal.

The patient attributed the cause of these muscular spasms to a violent fright which he had five years previous at the bursting of a circular saw, and which affected him so thoroughly that he became "purely white" in his face, and at the same time he began to appreciate a sensation of weakness in his whole body; not until fifteen minutes had elapsed was he able to recover himself to any degree. At that accident, however, he received no injury or mechanical shaking up of his body.

About 14 days afterwards the patient noticed these spasms for the first time, and they grew worse later on, but they did not disturb him at his work. Besides that the patient said that he felt the combined sensations of pain and pressure in the arms and legs a number of years before that fright, always, however, only during rest, never during his work or any other active movements.

There was no hereditary predisposition to neurosis.

The patient during his double stay at the clinic was treated with liver oil, with special consideration of his lung disease and in behalf of his improvement of nutrition through which a favorable effect was intended, although the muscular affection remained the same. During the month of March, after only a few galvanizations undertaken by Prof. Schultze, the spasms rapidly began to diminish in intensity, and after a number of days disappeared without leaving a trace.

The increase in the skin and patella reflex diminished in quick succession, so that by the end of April the normal relations were restored in this respect also. The patient was discharged from the clinic by the middle of May.

# CHRONIC PROGRESSIVE HEMIPLEGIA, WITH REMARKS ON TWO CASES OF UNILATERAL PARALYSIS AGITANS WITHOUT TREMOR.[1]

By Hugh T. Patrick, M.D., Chicago.

M. K., eighteen years old, of German parentage, was first seen September 20, 1903. The family history is negative, both parents are living and well. She is the last of five children, there having been one miscarriage eleven years after her birth. The first three children died of diphtheria. The fourth child, born two years after the third, is living, three years older than my patient, and is strong but nervous. The patient was born at full term after a normal labor, walked at eleven months and talked at eighteen months, but not plainly until five years old. Excepting the ordinary diseases of childhood, from which she made perfect recoveries, she was well, strong and active until the age of thirteen or fourteen. Both the patient and her mother are emphatic in their statement that she had perfect use of all extremities until the beginning of the present trouble. She has never had convulsions, has developed mentally in a normal way, was bright as a school child, a rapid writer, and acquired unusual skill at fancy work. Menstruation began at eleven years, has always been regular and also painless until about a year and a half ago, when she had been working very hard. Since then there has been some but not extreme dysmenorrhea. At about thirteen she began to be rather irritable, impatient and emotional, as is the elder sister, and the mother attributes the change to the influence of this sister.

Shortly before the age of fourteen years a slight clumsiness of the right foot was noticed and writing began to cause fatigue of the forearm. After a few months the limp had become very apparent, the child was no longer able to run well and disability of the hand was distinct. At the age of fourteen years and three months, six to nine months after the onset, the patient left school, and she remembers that at that time she could still write well and do fancy work well, though with considerable effort. Gradually the weakness on the right side increased, but when fifteen years old she was still well enough to take a place as domestic with some acquaintances. She states that at this time she could carry a child on her right arm, and could use the right hand pretty well in washing and drying dishes, but was quite unequal to the task of buttoning her clothes or those of the children. Steadily the disability grew and although her employers were unusually considerate, she was obliged to give up the place and go home in the spring of 1902, when seventeen and a half years old. Since that time her condition seems to have changed very little except as regards some symptoms apparently functional. As before noted, dysmenorrhea appeared just before she came home, and at the same time she became more irritable and emotional.

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[1] Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

Soon afterward she began to notice that often on rising the neck was swollen, but would return to normal in an hour or two. This was not a mere sensation, as the mother says it was plainly visible. Indeed, this transient swelling, apparently of the thyroid gland, continues, and for the last three or four months the right side has remained slightly enlarged. At times the face flushes smartly, and at such times she has headache. These flushes are sometimes, not always, coincident with menstruation.

During the whole course of the trouble there has been no pain whatever, no tremor, jerking or convulsions, no dizziness, tinnitus or vomiting, no affection of vision, speech or mentality, no involvement of the sphincters, and the general health has remained good.

She is at present a ruddy, well-grown, well-developed woman, five feet five inches tall, and weighs 140 pounds. Mentally she is normal. Results of examination of thoracic and abdominal viscera, blood and urine were negative. The special senses, pupillary reactions, fundi oculorum and cranial nerves reveal nothing abnormal, except paresis of the seventh and motor root of the fifth as hereinafter mentioned. Nothing in the history or examination indicates inherited or acquired syphilis.

There is an incomplete right hemiplegia, and the patient states that it seems to her that sensation is not so good on that side. She also says that sometimes the right hand and leg feel numb, especially at the menstrual time, but repeated careful examinations have shown sensation to be perfectly normal in all its qualities, including the localization of sensations, the sense of position and of motion and stereognosis.

Voluntary movements of the right facial muscles are slightly less perfect than of the left. With the face at rest the right eyebrow is a little lower than the left, and a trifle of facial asymmetry is noticed, the right side being slightly smaller than the left. On raising the brows to wrinkle the forehead horizontally the left eyebrow rises a little higher, and in showing the teeth the left corner of the mouth is retracted more than the right. The eyes are closed equally, and no excess of associated movement of the right side can be demonstrated.

The muscles supplied by the motor root of the fifth nerve show more difference. When the patient bites the teeth strongly together, masseter and temporal on the right side can readily be felt to contract less powerfully than on the left. Biting a pencil, she makes noticeably smaller indentations with the right side. Indeed, the patient asserts that on this side she can bite my finger only a little harder than I am able to endure. The pterygoids are clearly involved. Movement of the lower jaw toward the right brings the space between the canine and bicuspid teeth to the middle line, while toward the left the space between the lateral incisor and canine reaches the same point. When an effort is made to protrude the inferior maxilla straight forward beyond the upper, it deviates to the right to the extent of about three-fourths the width of the upper incisor. In opening the mouth *ad maximum* the middle point of the jaw makes a curving deviation toward the right, curving back toward the middle line on closing, but even at rest the interincisor space, or median line, of the lower jaw is slightly to the right.

The right half of the tongue seems to me slightly smaller than the left, but it is not wrinkled or flabby, lies straight in the mouth, and is protruded straight. Neck muscles show no noticeable difference, except those going to the shoulder. The right shoulder is shrugged somewhat less powerfully than the left, and all movements at the shoulder joint are weaker on the right side. The arm can be elevated to an angle of about forty-five degrees above the horizontal. Flexion and extension of the forearm on the arm show more difference, pronation and supination and movements at the wrist are weaker still, while the greatest weakness is evident in the flexors and extensors of the fingers and in the small hand muscles. Grasp of the left hand is unusually good for a woman; with the right hand the dynamometer can be held, but the index cannot be made to move. Power to pinch between the extended fingers is nil between the index and middle, scarcely perceptible between middle and ring, somewhat better between the little and ring fingers. There is some power in the thumb, so that objects can be held after a fashion between thumb and forefinger. For instance, she can still manage to do some crochet work by holding the needle in the right hand and moving the work in the left.

In walking, the right arm is not carried flexed and pronated, as is so frequent in ordinary cerebral hemiplegia. It hangs by the side, and while it doesn't "dangle" vertically, as does the arm in advanced progressive muscular atrophy or poliomyelitis of the shoulder type, the hand does swing flabbily to and fro with every step, and gives one the general impression of flaccidity. The most pronounced feature of the gait is a decided limp on the right side. The gait is neither ataxic nor spastic, nor is there very marked "steppage," but foot-drop is plain and the outer border drops more than the inner, so that in the forward swing the foot is in the equino-varus position. The patient toes distinctly neither out nor in. The right foot is raised higher than the left, it is not swung around in the arc of a circle, the toe does not catch the floor, but the heel does not come down first. Watching the patient walk one would say that the right leg was shorter, but careful measurement shows a scant one half inch difference. In the lower extremity the gradation of paresis is much as in the upper. That is, in a general way, the proximal muscles are strongest, those toward the distal end the weakest. Standing on the right leg the patient can flex the knee a little and straighten it again if she is steadied. Dorsal flexion of the foot is practically nil. With the patient recumbent the point of the foot can be moved upward not at all, but the toes can be very slightly extended. The calf muscles are good, but below normal. Standing on the right foot alone she can just manage, when steadied, to raise the heel from the floor. Inversion and eversion of the foot are impossible. Flexion of the toes is possible but feeble.

Aside from this deterioration of power toward the ends of the limbs, there is no localized paralysis; even no great preponderance in relation to physiological groups excepting as between the front and back of the leg. Flexors, extensors, rotators, adductors and abductors seem to be about equally involved.

The same applies to muscular volume. In no muscle or group of muscles is there unmistakable atrophy, but arm and leg on the right side are smaller in all their parts. The measurements in inches are: Arm, right, 10¾; left, 12; flexed, right, 12; left, 12¾; forearm, right, 9¾; left, 10¾; three inches above wrist, right, 7; left, 8; hand, right, 6¾; left, 7¾; over knuckles, right, 6¾; left, 7½; calf, right, 13⅞; left, 15⅜; foot-arch, right 8¾; left, 9⅛; foot-ball, right, 8½; left 9; thigh, six inches above patella, right, 20⅜; left, 22⅛. Trapezius, deltoid and scapular muscles are also perceptibly smaller on the right. These differences are not such as are ordinarily found in anterior horn and peripheral disease, and, considering that the patient was less than fourteen when the trouble began, it seems reasonable to attribute them, at least in part, to lack of development. This hypothesis is supported by the discrepancy in the hands around the knuckles (¾ inch) and in the feet (⅜ and ½ inch), where there is very little muscular tissue. Skiagraphs also lend support to this view as they show the bones of the right hand to be smaller than those of the left. The electric examination indicates that lack of development would hardly explain all of the difference. While there is no reaction of degeneration nor even partial reaction of degeneration, there is certainly more change than is found in the cerebral palsies of children, lateral sclerosis or multiple sclerosis;

even more than I have ever been able to detect in cerebral hemiplegia with muscular wasting—so-called cerebral atrophy. The muscles showing the greatest diminution in size are the small hand muscles, the tibialis anticus and the peronei, and in these there is quite marked quantitative change.

Despite the general appearance of flaccidity the pyramidal tract evidently bears the brunt of the lesion. All the deep reflexes are greatly exaggerated on the right side, including the scapulo-humeral and McCarthy's reflexes. It is even easy to demonstrate that the right masseter jerk is greater than the left, and a short-lived supinator clonus is easily elicited. There is only an indication of ankle clonus, but Babinski's extensor response is typical, and may be obtained by pinching the leg as high as the middle of the calf. All reflexes on the left side are normal. Contractures are absent, with the exception of some slight shortening of the calf muscles. The foot may easily be brought to a right angle with the leg, but to get it much beyond this requires considerable force, and even strenuous pushing will not dorsally flex it to the extent that can easily be accomplished on the normal side. None of the excess of associated movement, so generally present in the cerebral palsies of childhood, can be found.

There is no trace of incoordination or tremor of any kind; no fibrillary twitching, no nystagmus, no speech defect, no dysphagia. Phonation is normal, and laryngoscopic examination shows normal movements of the vocal cords. As before stated, sensation is normal.

The apparent flaccidity, as shown by the dangling arm, foot-drop and moderate steppage, and the presence of real spasticity as shown by great exaggeration of the reflexes and Babinski's sign, present a very peculiar clinical picture. The case is entirely unique in my experience and an inconsiderable search of the literature has disclosed nothing just like it. Progressive muscular atrophy, amyotrophic lateral sclerosis, syringomyelia, multiple sclerosis, muscular dystrophy, neuritis, spinal tumor and hysteria may easily be excluded. It is difficult to imagine any gross cerebral lesion which could cause all the symptoms of this case and yet cause no sensory disturbance, no headache, vomiting, dizziness or optic neuritis, no more involvement of cranial nerves, no spasm, twitching, tremor or incoordination, no interference with speech and no mental deterioration. Degeneration of the upper motor neurones would seem best to explain the condition, but there is not nearly so much spasticity as in primary lateral sclerosis, and there is more diminution in muscular volume than I have seen in cases of that disease. I am unwilling to make a diagnosis. Perhaps I would better say I am unable, but I think the case more closely conforms to the clinical type proposed by Dr. Charles K. Mills than to any form of disease with which I am acquainted, and his hypothesis of pathology, "a slowly increasing degeneration of the pyramidal fasciculi or of the cerebral motor neurone system," seems to suit the symptoms of my patient.

About three years ago Dr. Mills[2] reported a case which he called unilateral progressive ascending paralysis, and which he thought probably represented a new form of degenerative disease. As he stated in that report, the patient had been sent to him by me, and when I first saw the case just described, my first thought was that I had to do with a second example of the same affection. His patient I had not seen since July, 1900, eight months after his visit to Dr. Mills. To my written request for a further examination the patient quickly responded, and on reexamination I made a diagnosis quite different from that of Dr. Mills. My only surprise is that I had not thought of it before. I believe the man to be suffering from paralysis agitans, unilateral as to distribution (the so-called hemiplegic form), and without tremor. I believe this to be correct because all the symptoms except an attack of something like herpes can be explained by this diagnosis, and because I have now under observation another case almost exactly like it, in which the tremor is beginning to develop. Briefly, the case is as follows, my description agreeing essentially with that of Dr. Mills.

At the time of his first visit to me, September 26, 1899, M. J. P. was fifty-two years old, and the trouble had begun twenty months before. His wife first noticed that his right foot occasionally caught the floor or rugs, and before long he noticed it himself and made a conscious effort to lift that foot better in order not to stumble. After about eight months he was conscious of a dull ache in the extremity, and a few months later was sure that the right leg was not only more clumsy, but was weaker than the left—at any rate it felt weaker and was more easily fatigued. Five months before coming to me (fifteen months after the inception) he became aware that he carried the right arm against the side and crooked at the elbow, and soon afterward it began to grow a little stiff and was filled with a dull, heavy ache. Why this in itself did not suggest the diagnosis to me I am unable to comprehend, because only a year and a half before I had seen a gentleman sixty years old who complained of exactly the same thing (the leg being unaffected), which case I at once recognized as paralysis agitans, *sine agitatione,* a diagnosis confirmed by the subsequent appearance of all the classical signs. With the stiffness (in the case now under consideration) came some disability in the fingers. In writing the patient grasped the pen too firmly, and in a few minutes hand and fingers would grow quite stiff and the arm would ache so that he could not continue. Buttoning clothing and other finger acts became difficult. Since that time the disability of the hand has been more prominent, at least more annoying than that of the leg. It has also been more progressive. Indeed, the patient affirms that the lower extremity is about as good now (April, 1903) as it was three years ago.

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[2] Mills. Journal of Nervous and Mental Disease, April, 1900.

At the time of my first examination no record of measurements was made, but I noted the patient's statement that eight months previously the right thigh had been found to be one inch smaller and the calf one-half inch smaller than the left. Dr. Mills found a difference of one and one-fourth, and five-eighths respectively, and eight months later I found the differences to be one inch and five-eighths inch. The upper extremity showed no wasting. Aside from some increase in the disability of the hand, the present condition is so nearly that of three and a half years ago that the same description may serve for both, with such small exceptions as will be noted.

As regards the lower extremity, it is first to be noted that the disability is more apparent than real. To the patient there is a feeling of stiffness and clumsiness; to the observer the leg in action looks rather stiff and clumsy, but there is no spasticity, a scarcely perceptible paresis and no incoördination. On passive motion there is a little more resistance on the right side than on the left, but it is no more marked in quick or sudden movements than in slow ones—a nice distinction between the rigidity of paralysis agitans and that of pyramidal tract disease. The patient can stand on the left foot and easily pass the right over an object twenty-six inches high. He can run at fairly good speed, the only peculiarity being that he raises the right foot a little higher, and has not the same spring on that side; even hopping is well executed. He can stand on the right foot alone and let himself down until the thigh is at an angle of about forty-five degrees, and then rise so that he stands erect on the one leg, and he can stand on the right toes or on the heel. Especially the last I consider to be a severe test. Thigh measurements eight inches above the patella are eighteen and a half and nineteen and a half inches; calves, twelve and five-eighths and thirteen and one-fourth. In other words, the difference now is what it was more than four years ago.

Since the beginning, arm and hand movements have steadily but gradually progressed for the worse. The fingers are more clumsy, the arm and hand are weaker, the movements of even the larger joints less free. Putting the hand into the hip or even side pocket of his trousers is a slow matter and requires some effort. To button the collar behind is almost impossible, and buttoning it in front is no easy matter. For six months he has been unable to button on the left cuff. The shoulder muscles are all strong, perhaps fractionally weaker than on the other side; the two biceps muscles seem to be equal, the right triceps not quite so strong as the left. Pronation and supination of the forearm are equal on the two sides, as are flexion and extension at the wrist. At the time of Dr. Mills' examination the grasp of the right hand was stronger than that of the left (180—160), now the right is weaker (85—95).* Adduction and abduction of the fingers are slightly weaker on the right. As late as July, 1900, the right arm and forearm were slightly larger than the left; now the right arm is one-fourth inch smaller than the left; the forearms are equal. The same quick fatigue of the arm and aching on exertion remain. But the great discrepancy between the two hands is not in strength, but in freedom and promptness of movement—the disability characteristic of paralysis agitans. Apparently there is no movement of the fingers that cannot be executed, but no movement can be done quickly. For instance, movements of the fingers as in playing the piano can be executed with sufficient amplitude but only with extreme slowness. Smallest objects can be picked up with certainty but not quickly. This deliberateness of movement applies as well to muscles of the arm. Alternate flexion and extension at the wrist cannot be done rapidly, although as just noted, the strength is normal. The tooth brush is now used in the left hand, because with the right he cannot make the usual quickly alternating movements at the wrist. It is impossible for the patient to make a sudden movement, such as one would make in attempting to catch a fly. On command, a rectangular movement is very much slower on the right side. That is, if told to swiftly raise the hand from the knee ten or twelve inches, and then carry it ten or twelve inches horizontally one way or the other, the starting, the change of direction and the stopping take place slowly. This lack of prompt response to volition is less evident in movements at the shoulder. Last summer the patient played golf and could do so fairly well, but his swing was rather slow.

Movements of the face seem to be slightly better on the left side but the difference is so small as to be indeterminate.** The frontalis acts equally on the two sides, but after some seconds of continuous action the right side tires a little, and the eyebrow drops a trifle. Masseters, temporals and pterygoids are equal.

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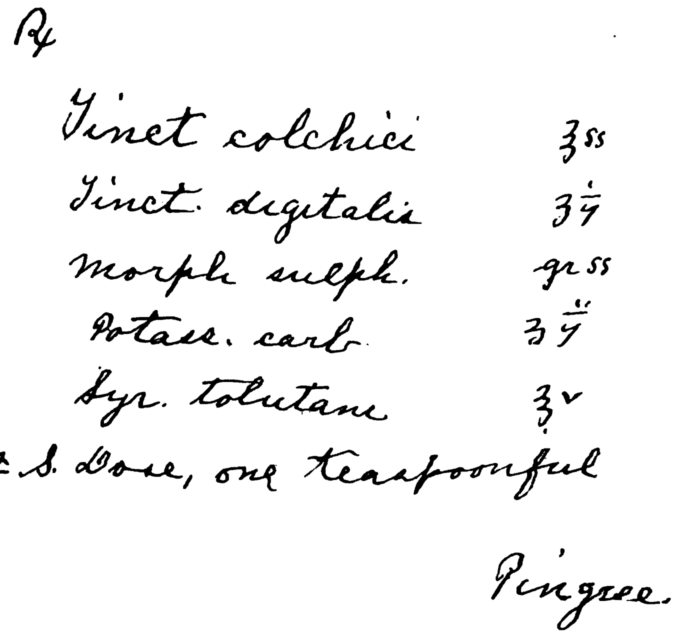
*My dynamometer is evidently much stiffer than that of Dr. Mills.

**Since reading this paper I have examined a case of unilateral paralysis agitans in which the facial difference was much greater than in this one.

Achilles-jerk, knee-jerk, wrist-jerk and triceps-jerk are all brisker on the right side. An evanescent trace of ankle clonus can sometimes be elicited. Dr. Mills found patellar clonus. Apparently this varies as I have been unable to get it. The jaw-jerk is absent, as is Babinski's sign.

While comparing the muscular strength of the arms I was suddenly struck by a phenomenon that seemed to present the key of the situation. On demand the patient was forcibly supinating the left fore-arm against resistance, when I noticed that the right hand had assumed the position typical of paralysis agitans, and was shaken by a rhythmic tremor like that of this disease. The motion of this tremor seemed to be largely rotatory, that is, of pronation and supination. On retesting in the same way, the tremor was not so pronounced, but the motion was more a flexion and extension of the fingers, like that so often seen in paralysis agitans. I afterward noticed that the fingers are habitually carried in the position of paralysis agitans, though without tremor. For a long time, the patient does not know just how long, exertion of the right hand or arm has caused not only an aching pain but also tremor. This has been particularly evident during and



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Tinct colchici ℥ss
Tinct. digitalis ʒi
Morph sulph. gr ss
Potass. carb ʒii
Syr. tolutani ℥v
M. S. Dose, one teaspoonful

Pingree.

Fig. 1. Written slowly and carefully.

after some strain, as in buttoning the collar behind. At the present time this is very easily elicited by attempts to move the fingers rapidly as in playing the piano, and this tremor, though transient, is indistinguishable from that of Parkinson's disease. When the patient lies on the back and holds the right leg in a vertical position, it soon begins to ache, and is shaken with a rhythmic tremor affecting principally the quadriceps extensor. On the left side the same maneuver causes less tremor beginning later. When the patient forcibly raises the naso-labial folds and wrinkles the nose, there is an almost rhythmic tremor or jerking on the right side, the rate being about six per second. Tremor is quite evident in writing. If he takes plenty of time his penmanship is very good, but shows tremor in numerous letters. (Figure 1). If he tries to write rapidly, tremor is so bad as to make the product almost illegible (Figure 2). For several years a very annoying symptom has been a feeling of stiffness and drawing below the right occipital region, or in the right upper neck, leading the patient to frequently rub the place and forcibly move the head about in an effort to relieve it. Eighteen months ago the palm of the right hand began to perspire in excess. It is always moist, and last summer it often dripped sweat. Elsewhere there is no excessive perspiration. Lately, the patient has noticed that the skin on the back of his right hand is smoother, does not present the distinct reticulation seen on the other hand; looks more like parchment.

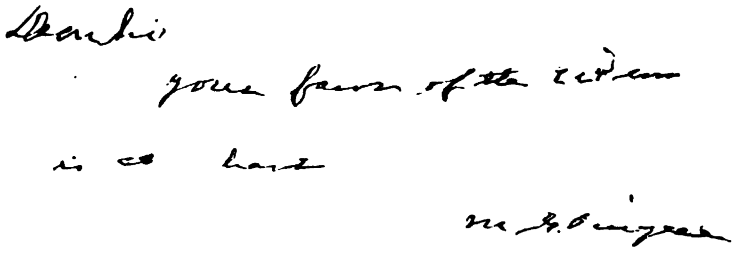

Fig. 2. Written rapidly.

Sensation, coördination, stereognosis, special senses, fundi oculorum, mentality and speech are normal. There is no fibrillary twitching, intention tremor or nystagmus. Pain, headache, dizziness, vomiting, tinnitus and paresthesia have been absent. Sphincters are good and sexual power intact. During the last year a curious sequel of intercourse has been observed. Beginning ten or fifteen seconds after the termination of coitus, the right leg becomes rigid in extension, and is seized with a rapid tremor or clonus lasting about fifteen seconds. This is unaccompanied by any pain like that of muscular cramp.

On January 18, 1903, the patient was put on 1-100 grain hyoscine hydrobromate *t.i.d.* Eight days later he reported that he was sleeping better, that the tense feeling in the neck was gone, and that the right side felt less clumsy. He could wrinkle the nose without the tremor formerly noticed, and forced supination of the left forearm, while causing the fingers of the right hand to assume the typical Parkinson position, induced no tremor Writing was easier and caused less discomfort and tremor.

For purposes of comparison I wish to report in brief the following case:

T. K., a merchant, fifty-four years old, was referred to me December 30, 1901. Except the fact of rheumatism in the mother, the family history is negative. At intervals for twenty or thirty years the patient has had lumbago. Once it lasted two or three months. Twenty-five years ago he had rheumatism in the right ankle for some days. He has been somewhat troubled with hemorrhoids, is constipated, has taken a laxative every night for four years.

A year and a half before my examination he began to have pain about the middle of the right arm whenever he put the hand into his hip pocket, sometimes on other backward movements. This lasted for ten months, from May, 1900, to March, 1901, and during part of this time the middle joint of the right index finger was swollen and tender. During the following summer, 1901, the right upper extremity became weaker—as the patient expressed it. That is, in writing the hand soon became fatigued, and in rowing the right arm and shoulder tired before the left. Like the preceding patient, he began to carry the arm semi-flexed, and it felt rather rigid and clumsy. The disability gradually increased, writing became more difficult, and caused a tired ache in the arm and shoulder. A very short time before coming to me the right knee had begun to feel slightly rigid, and for a few days he had been annoyed by the heel occasionally catching the ground. In the upper part of the neck on the right side was a distressing sensation like a drawing or pressure.

Examination revealed a state of affairs very much like that of the preceding case: On the right side a plastic rigidity, unlike that of pyramidal tract disease, like that of paralysis agitans; slowness and clumsiness of movement, no real paralysis; the right extremities slightly smaller than the left, and the deep reflexes of the right leg more active than those of the left. There was no tremor except after some physical exercise, when the whole right arm trembled. The fingers of the right hand were habitually carried in the position typical of paralysis agitans, and he presented the characteristic facies of this disease.

Since that examination his condition has changed very little, except that the leg is slightly more involved, the foot catches the ground more frequently, and he walks with a slight "hitch" on the right side. He left his business and has taken excellent care of himself. Beginning about December 1, 1902, he has noticed that the left elbow seems rather stiff and a little sore, especially when the arm has been for a long time in the same position. Strong efforts at pronation, supination, or flexion of the left arm against resistance cause an accentuation of the Parkinson attitude of the right hand and generally some tremor.

In this case there would seem to be no cause to doubt the correctness of the diagnosis, paralysis agitans. But excepting the facies, the other case, the one examined by Dr. Mills, presents almost exactly the same symptom-group: the same ache and fatigue on exercise, the same slowness and clumsiness of movement without paralysis and without ataxia, the same way of carrying the arm, the same position of the fingers, the same sort of discomfort in the suboccipital region on the affected side, the same sort of tremor induced in the same way, the increase of deep reflexes and diminution of muscular volume on the affected side. That one began in the leg and the other in the arm seems to me to be important. I agree with Dr. Mills that the attack of more severe pain and skin eruption (herpes?) of the first case does not constitute an essential feature. Unilateral distribution of paralysis agitans is the rule in the incipiency. Marked unilateral paralysis agitans without tremor is unusual, but it is far from being unrecognized in the literature. A greater difficulty in these two cases, especially in the first one, M. J. P., is the combination of diminished muscular volume and increased reflexes. After all, the former symptom is very inconsiderable in degree, and while the latter is not the rule in paralysis agitans it is not rare. In my opinion, lack of use is too often proposed to explain reduction in size of an extremity, and I am not prepared at present to assert that this will account for the difference of one inch in the thighs and five-eighths of an inch in the calves, but I do think that it is quite as reasonable to thus explain it, or to simply say that it is an unusual manifestation of paralysis agitans as it is to invoke the aid of a new and not yet understood disease. Indeed, Jolly[3] speaks of the diminution of muscular volume which ordinarily appears in the course of paralysis agitans. Besides, degeneration of the pyramidal tract alone would explain it no better. The appearance of tremor during or immediately after exertion, and in the involved but not trembling extremity on forced activity of the other side, are manifestations of paralysis agitans often noticed in incipient cases or old ones without tremor.

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[3] Jolly: Handbuch of Ebstein and Schwalbe, Vol. IV, p. 865.

Ten months after Dr. Mills' report Dr. Spiller[4] published another case under the same title, but I am inclined to think that the disease is not the same. His case showed distinct spasticity, ankle-clonus and Babinski's sign, but was peculiar in presenting optic atrophy on the affected side. Furthermore, the patient occasionally found some difficulty in retaining the feces. Judging from his report I should say that he probably had to do with some affection different from any of the cases in this report.

To summarize, I would say that my first case apparently belongs to the clinical type indicated by Dr. Mills, but that my second case, the one reported by him, certainly affords no basis whatever for the creation of a new type, as it is an example of unilateral paralysis agitans without tremor.

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[4] Spiller: "A Case of Unilateral, Progressive, Ascending Paralysis." Philadelphia Medical Journal, February 9, 1901.

# THE WORK OF A NEUROLOGICAL DISPENSARY CLINIC*

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During the year 1902 there were 1,780 new patients suffering from some well-defined form of nervous disease who presented themselves for treatment at the Vanderbilt Clinic. Of these 953 were females and 827 were males. This number does not, however, represent all of the new cases applying to the clinic for treatment. This number was 2,044, 1,068 being women and 976 men. At least 134 of these did not present symptoms referable to the nervous system, and in 130 patients no definite diagnosis could be made. The total number of visits made by these patients was 10,121, thus making an average of about five visits per patient. Inasmuch as many of the patients come once only it is probable that the average number of visits is between seven and eight. A number of patients came once a week throughout the year. In this connection it is of interest to note that a few patients suffering from slowly progressive affections have been coming to the clinic almost monthly ever since the clinic was opened in 1889.

Contrasting these figures with those of the first year of the clinic's opening, it is found that the number of patients who have come for treatment has been doubled. Thus in 1888-1889 there were 944 patients or, for the first twenty-three months of the clinic's opening, there were 1,888 cases, less by 200 than the number for the twelve months of 1902 alone.

*Mental Diseases.*—In attempting to present an analysis of this material we have deemed it of interest to consider at first the purely mental diseases that have come for diagnosis and treatment. The commonest forms of mental affection presenting themselves have naturally been in children. In these deficient development is represented by idiocy in six cases, imbecility in 54, making a total of 60 cases or an incidence of about three per cent.

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*Report of Clinic of Dr. M. Allen Starr, 1902.

It may also be of interest to note here that the Board of Education of this city has already taken steps to establish supplementary schools for backward children (called secondary schools on the continent of Europe). A few schools for this class are in successful operation, being maintained at other hours in the same school building as the regular schools. Obviously some systematic examination should be made in every child dismissed from the regular school system by school teachers or medical inspectors because of its mental deficiency. Such examination should be made by properly qualified physicians appointed by the Board of Education, or, as Dr. Starr suggests, the city might be divided into districts and the schools refer their cases to the nearest neurological clinic. As to whatever arrangements are employed all agree upon the necessity of supplemental schools, and the importance of systems of medico-pedagogical training for the defective.

The insanities are not well represented in dispensary work. In men, general paresis was the commonest affection, 23 (one per cent) presented unquestioned evidence of this affection, and as would be expected, always in the early stages. There were no women with paresis, which seems rather peculiar, inasmuch as syphilis, the most important etiological factor of paresis, is almost equally common in women, particularly in the lower strata of society. Probably the less exacting symptomatology for general paresis in the past few years accounts for the increased number of cases for this year's statistics, and for the apparent increase of the disease in general during the past decade. At present the signs of cerebral syphilis, plus positive evidence of mental alienation, are considered by many as sufficient data for an early diagnosis of general paresis.

Taking into account the essentially systematized and persecutory nature of the fixed delusions in paranoia, and the personal well-being of such patients, it is surprising that as many as seven men and one woman presented themselves at a neurological clinic. In most instances the patients come to the clinic for the secondary development of distressing hallucinations of hearing. The occurrence of melancholia in six men and in 24 women should be considered in the light of the older classification of the disease and not in the restricted sense of Kraepelin as a type of insanity of the involution period. In other words it includes manic-depression as well as true melancholia.

Senile dementia was diagnosticated in three cases. There was one patient recorded suffering from mild puerperal mania.

Cerebral gumma, and cerebral syphilis with mental symptoms, were diagnosticated in six instances. Therefore, the total number of patients with mental trouble was 131, or seven per cent. This is not a great number in a large neurological clinic when one recalls that patients suffering from mental disorders prefer to visit more or less voluntarily a clinic of this type, which, of course, does not possess detention wards or even hospital wards of any sort, a feature which should not be lost sight of in establishing out-clinics with psychopathic hospitals in close connection with large neurological dispensaries.

*Nervous Diseases (Functional)*.—Dividing the neurological cases into those usually classed as functional and those organic, there were 873 (49 per cent) of the former, and 776 (44 per cent) of the latter, distributed as the following brief record indicates.

Neurasthenia is par excellence the disease of dispensary patients. A more complete analysis of these cases is contemplated in a further communication. Here it may be said that there were 416 (23 per cent) patients who presented themselves for treatment, in whom this affection was diagnosticated, 250 in men and 166 in women. Apart from the marked sexual element in 62 male cases and 38 menopause neuroses, the cases of neurasthenia presented no particularly interesting features. The foreign population contributed over eight-tenths of the neurasthenics. Among men the Russian Jews preponderated.

Hysteria is given as the diagnosis in 80 cases. There was a history of major hysteria in but three cases. The remainder represented badly trained individuals of unstable nervous organization.

The *convulsive disorders* were represented by 135 (19.5 per cent) cases of chorea, of which 128 were of the usual type of this disease. There were five cases of chronic chorea and two patients with hemichorea, i.e., cases of chorea of the latter class which remained persistently unilateral.

Epilepsy, next to neurasthenia, contributed the largest incidence with 180 cases (ten per cent). An analysis of these cases is contemplated elsewhere.

Convulsive tics were not common, there being only 24 cases, about one per cent, of which 15 were of the ordinary type of spasmodic tic. Seven were obviously of the early type, and six were of tic douloureux. Women preponderated in the ratio of two to one.

There were 15 stammerers. The proportion of stammering in the general population would from these figures seem to be very small, but in view of the fact that we have no definite means of determining the general proportion of stammerers, some figures as recently published by M. Chervin of Paris in a monograph may be of interest here. As stammering is a cause of exemption from military service, it is possible to determine its frequency in different continental nations. It appears that of 1,000 conscripts examined the number rejected is as follows:

| | Per 1,000 |
|---|---|
| France | 7.50 |
| Switzerland | 3.23 |
| England | 2.87 |
| Austria-Hungary | 2.20 |
| Belgium | 2.10 |
| Italy | 0.86 |
| Russia | 0.19 |

Comparing these numbers with those that represent the frequency of nervous affections, he finds that in France the nervous affections are exactly twice as numerous as the cases of stammering; in Belgium they are four times more numerous, in Italy seven times, in Switzerland eight times, and in Russia thirteen times. No statistics of stammerers in the United States are given.

There were three patients with motor speech defects, and three patients with simple idiopathic tremor.

Paralysis agitans showed 17 cases, eight men and nine women. They did not present any features apart from the usual types.

The etiological statistics of the disease throw no new light on its pathogenesis.

*Organic Lesions* (*Peripheral Nerves*).—The peripheral nerve lesions of one type or another were numerous. They represented 379 cases (21 per cent), distributed equally between neuralgias 145 cases, neuritis 117 cases and peripheral palsies 117. Of this large number of peripheral nerve cases men were affected in 244 and women in 135 cases, showing the greater incidence of men to this type of lesion.

Of the neuralgias, sciatica was the most common, 48 cases; supraorbital in 16 (9 male and 7 female); intercostal 14 (3 male, 11 female); facial 25 (15 male, 10 female); generalized neuralgia in 18 (13 male, 5 female); brachial in one, coccygeal in one, ulnar one.

Neuritis was general in 12, traumatic in 18, lead in 11, alcohol in 7, occupation in 8, arthritic in 6, diphtheria in 3, typhoid in 1, circumflex 1, ulnar 1.

Peripheral palsies were represented by 117 cases. Facial palsies were most common in 41 (17 male, 24 female); musculospiral in 35 (29-6). Erb's palsy was represented by 5 cases, the ulnar nerve in 5, circumflex in 1, lead palsy 5, larynx 1.

Affections of the spino-peripheral neurone were present in but 46 cases. Of these anterior poliomyelitis was diagnosed in 24 cases. With reference to the incidence of these cases, 16 cases showed an initial history of fever, headache and vomiting. There was a history of initial convulsions in one case, and no recorded histories in seven. On the question of causation the history is negative in 16 cases, and given as due to a fall, measles, infections, dentition and pneumonia in 1 case each. The months of invasion are of interest. There was 1 case in January, none in February, 1 in March, none in April or May, 1 in June, 6 in July, 8 in August, 2 in September, none in October or November, 1 in December and no date given in 4.

Chronic anterior poliomyelitis was present in six cases, presenting the picture of progressive muscular atrophy. These were all in men.

Ophthalmoplegia was present in 11 cases. These patients were all men.

Tabes dorsalis was present in 32 patients. Only two of these were women.

Of these 32, record of 25 was kept—all males. The following occupations were represented: one each, driver, carpenter, farmer, soldier, bookkeeper, tailor, stone-cutter, fireman, janitor, butcher, lithographer, sleigh-maker, musician, cigar maker, butler; 2 barkeepers, 3 salesmen, 2 cooks, 2 laborers. The earliest age at onset was 28, the latest 49, although one patient is put down as 14 years of age. It is doubtful if this early case is one of tabes. The average age of this series of 25 is about 35 years.

Bearing on the question of etiology, it was found that 14 had had syphilis, two had led extremely dissipated lives and nine presented no evidences nor history of syphilis, nor other etiological factor. In ten of the 14 presenting syphilis the initial lesion had been acquired 20, 8, 10, 28, 26, 18, 13, 30, 15, and 19 years previously, respectively. No special hereditary factors were present in any. In one there was senile dementia, in another grand mal epilepsy, in another tuberculosis, in the parents.

A few interesting facts concerning the earliest symptoms may be recorded. There were, as earliest symptoms, neuralgic pains in 11, perforating ulcer in 1, gastric crises in four, general weakness in the legs in 2, blindness in 4, ataxia in 3, and myosis in 1. The knee-jerks were absolutely lost in 24 and diminished in 1. The Argyll-Robertson pupil was present in 21 instances. There was normal light reaction in 3; myosis and optic atrophy in 1. Other detailed analyses are left for a special paper.

Friedreich's ataxia was found in 3 patients, 2 men and 1 woman.

Lateral sclerosis was present in 6 men and one woman.

Amyotrophic lateral sclerosis was present in 3 cases.

Multiple sclerosis was found in 6 men and in 4 women.

Myelitis was present in only two cases, certainly bearing out the recent contentions of Starr, Gowers and Risien Russell, that myelitis is really a rare affection.

Ataxia paraplegia was present in 5 men, and spastic paraplegia in 2 men.

Tumors of the spinal cord were present in two patients, one man and one woman.

*Brain Affections.*—Organic diseases of the brain were observed in 85 cases. Eighteen of these were in children presenting the symptoms of infantile cerebral palsies. Causation is here represented as whooping-cough in one, trauma in two, prolonged labor in four; the rest were negative. One patient was diplegic. Monoplegia was present in two men and two women. Hemiplegia was present in 58 patients, an incidence of 2.7 per cent. Forty-three in men and 15 in women. Two patients had aphasia. Tumor of the brain was present in four cases, three men and one woman; two cases of cerebral gumma were diagnosed.

Syphilis of the brain was diagnosed in 12 cases (3 men, 9 women).

*Vascular Lesions.*—Vasomotor affections were diagnosed in 16 patients, angioneurotic in 4, flushings in 3 and erythromelalgic symptoms in 9 patients, 6 men and 3 women.

Exophthalmic goiter was present in 8 women.

*Miscellany.*—Alcoholism in 43 males, 1 female; muscular dystrophy, 3 males, 1 female; atheroma 3; arteriosclerosis 12; concussion of brain 3; fracture of skull 1; headache, 10 males, 75 females; hemicrania 2; herpes 1; hemianopsia 1; insomnia 2 males, 11 females; pachymeningitis 4 males, 1 female; optic atrophy 2; paresthesiæ 5 males, 11 females; senility 1; incontinent bladder 1; local atrophy 1.

# Society Proceedings.

## THE PHILADELPHIA NEUROLOGICAL SOCIETY.

April 28, 1903.

The President, Dr. H. A. Hare, in the chair.

*Poliomyelitis in the Adult Involving All Four Extremities.*—Dr. John K. Mitchell exhibited this case. C. R. Wadley, aged twenty. There is nothing in the history, either personal or family, of interest. The boy was in ordinary health and has never suffered any injury until August 15, 1901, when he suddenly suffered for a day or two with stiff neck and pain in the back. Two days later he lost consciousness, according to the account which he has given, and remained unconscious for twenty-four hours. He had fever for a week following, but no vomiting, diarrhea, or convulsions.

On recovery from the fever he was completely paralyzed and could not move his head. Sensation remained perfect. Motion gradually returned, first in the head, then in the upper right arm, then in the lower left arm, then in the upper left leg and then in the lower right leg.

In the autumn of 1902 he was admitted to the Orthopedic Hospital. He was then entirely unable to stand. He could use his hands very little and could not raise his legs from the bed. When he endeavored to sit up, he was unable to get his spine straight from weakness in the back muscles. The worst parts at that time were the lower right arm and hand, the upper left arm, the upper right leg and the lower left leg. The affected parts were very cold and presented slight contractures. He had no knee-jerk and the plantar reflex was very small, cremasteric reflexes were absent and the abdominal slight. There was fair reaction to faradism in the arms; very poor in the legs. There was no degenerative reaction, KCL greater than AnCL everywhere.

He has improved very slowly, but steadily, and the only member which seems unsusceptible of betterment is the right hand, in which the destruction of the intrinsic muscles has been so complete that he has very little use of it, and very little prospect of a valuable member. In every other way there is a change for the better, and the boy is able to walk, supported in the wheeled-crutch, and can take a few steps with assistance even without the degree of support which this apparatus gives.

The case was shown as an interesting example of widespread and irregular distribution of a poliomyelitic paralysis at an age when patients are commonly exempt.

Dr. A. A. Eshner referred to a typical case of anterior poliomyelitis in a boy seven or eight years of age involving all four extremities. At the same time he had under observation a boy five or six years of age with flaccid paralysis of the extremities who was at first thought to be suffering from anterior poliomyelitis, but who later exhibited derangement of speech and incoördination of movement which led Dr. Eshner to believe that the trouble was of cerebral origin.

Dr. H. A. Hare said that there had been admitted to his ward in the Jefferson Hospital, six or seven weeks ago, a woman about nineteen years of age, with the statement that she was convalescent from typhoid fever and was universally paralyzed. There was a history of five weeks' fever which careful questioning reduced to two weeks. The temperature had ranged as high as 105° F. When admitted she was absolutely paralyzed in the upper extremities and could slightly move one leg. There was no interference with speech or intellect. The hands presented a rhythmic tremor resembling that of chorea as it is seen in the dog. The emaciation was marked. There was exaggeration of the reflexes both at the elbow and at the knee, but not in the feet. Under careful nursing, massage and tonics the patient rapidly improved so that in ten days she could move all four extremities. At the end of that time she, unexpectedly, left the hospital and has been lost sight of.

Dr. John K. Mitchell believed that one reason why the limit of probable beginning improvement was within one or two years was because in bad cases the unaffected muscles atrophied from disuse. As showing that even very old cases are not insusceptible to improvement he referred to a case of fifteen years' standing in a girl eighteen years of age. When she came under observation there was almost complete paralysis of both legs. She was able with difficulty to shuffle across the floor a distance of thirty feet. She is now, after a year's treatment with massage and electricity, able to walk a mile with the aid of a stick.

*A Case of Brachial Monoplegia of Spinal Origin.*—This report was made by Drs. W. G. Spiller and T. H. Weisenberg. The patient had been in the service of Dr. Spiller at the Philadelphia Hospital. The clinical history was very incomplete, but there was sufficient to show that the man had had a paralysis of one upper limb. An area of sclerosis was found in the lower cervical region in the lateral column and anterior horn on the side corresponding to the paretic limb. This sclerosis was evidently the result of thickening of the blood vessels of the affected part. The case was important as an example of monoplegia of spinal origin in an adult.

*A Case of Thomsen's Disease, Associated with Pseudo-muscular Hypertrophy.*—This case was reported by Dr. Horace Carncross. The patient, a young man aged twenty-one years, was troubled with rigidity after resting. At birth he was a large child, weighing fifteen pounds. When nine months old he weighed forty-two pounds. When he was two years of age stiffness of the hands was noted and later of the legs and other parts of the body. If he fell he could not get up until after the lapse of five minutes. At this time he was very strong. At the age of six years he went to Girard College. From ten to fifteen years of age he was not as strong as before, or as his development would indicate. At the age of seventeen he began to waste. He has great difficulty in getting up in the morning. In going up stairs the first two or three steps give him much trouble, after that he goes up more easily, and at the second flight he can run. All the muscles are rigid, including those of the tongue.

The sensory symptoms are negative. The patellar reflex and biceps-reflex are absent, except on reinforcement. The electrical reactions are normal, except that the muscles respond sluggishly. The eye-grounds are normal, and the pupils react normally. The family history is negative, except that the mother had recurrent attacks of what were spoken of as facial paralysis, but which from the history were more probably attacks of facial spasm.

Dr. F. X. Dercum referred to a case which he had presented before the Society some years ago,—that of a bricklayer who when he started to work would suffer from a tonic spasm of the muscles. In that case there was no family history suggesting Thomsen's disease. He had merely termed it a case of myotonia. The present case he considered an anomalous one, but he thought that it was a muscular dystrophy associated with myotonia.

He believed that a study of these cases showed that our conceptions of the various types of dystrophy needed widening and readjustment.

Dr. D. J. McCarthy referred to three cases of Thomsen's disease which he had seen in Jolly's clinic in Berlin. In two of those cases the symptoms were no more marked than in the case shown tonight. The occurrence of facial spasm in the mother, and the fact that in the patient the condition had been present from birth, he considered of interest.

*A Note on Periodic Insanities with the Report of Three Cases of Intermittent Melancholia.*—Dr. Alfred Gordon read this paper.

Dr. F. X. Dercum said that mania, melancholia and circular insanity were essentially recurrent diseases. The practical point brought out by the paper was that irregular, intermittent melancholia was unfavorable as to prognosis. Ordinary melancholia yields a more favorable prognosis, but the attacks are liable to recur after rather prolonged intervals. Our suspicion should always be excited when the onset of melancholia is sudden and the recovery abrupt. It usually means recurrence after a relatively short interval as compared with ordinary melancholia.

Dr. John K. Mitchell referred to the fact that even in ordinary melancholia, recovery may occur suddenly. The line separating simple melancholia and the recurrent variety is very indistinct.

*Senile Dementia.*—This paper was read by Dr. Pickett.

Dr. F. X. Dercum regarded the present admirable communication as a worthy companion paper of Dr. Pickett's previous communication on dementia præcox. The paper was full of suggestions and brilliant generalizations. Some were quite startling, while others were in keeping with what we should expect.

Dr. Charles K. Mills said that with regard to the occurrence of other types of insanity than senile dementia in old age, he was quite sure that this was the case, and had seen a few striking illustrations. He referred to the case of a man eighty years of age who during his previous life had had at least five or six attacks of melancholia. He had retained his general mental vigor. At the age of eighty years he developed a typical attack of melancholia of the same type as he had previously had, although perhaps more severe.

# Periscope.

## CENTRALBLATT FUR NERVENHEILKUNDE UND PSYCHIATRIE

(15 January, 1903.)

1. On the Limits of Psychiatric Knowledge. R. Gaupp.
2. On a Peculiar Katatonic Symptom-Picture Removed by an Operation. K. Bonhoeffer.
3. Ophthalmoplegia Interna Traumatica. Ernst Schultze.
4. The Trend of Investigation in "Psychologic Studies." W. Weygandt.

1. *On the Limits of Psychiatric Knowledge.*—Gaupp says: Our science is not merely a branch of natural science, but the symptoms to be studied belong mainly to another field of labor, not entered by the explorer of natural science. It is a mistake to believe we can localize the psychical elements and thus make the psychical processes understood. Pathological histology will never do much for general psychopathology, especially not regarding the laws of psychical phenomena of healthy and sick persons. There is no possibility of understanding psychical events by their material bases, which we do not know in individual cases. Chemical science too cannot explain the psychical phenomena,—it being nothing else than molecular mechanics, which cannot be compared with psychics. The facts of anatomy and chemistry not helping in the investigation of psycho-pathological phenomena, our etiological knowledge is defective, and a difference of opinion arises regarding the axiom that like causes have like effects. It is said, for instance: Because one drinker suffers from delirium tremens, a second one from mania with jealousy, a third one from Korsakoff's psychosis, it cannot be that the same causes produce the same effects. However, not one cause alone, but several of them result in psychoses; we may recognize only one, and so this one may appear to have several effects. These difficulties lead to the conception of exogenous and endogenous causes of psychoses; some authors speak about predisposing and exciting causes. We have made some progress regarding the exogenous, but less about the endogenous causes. The expressions "degeneration" and "psychopathic disposition" show the uncertainty and vagueness of our opinions.

We know very little as to the individual psychological attributes of the predisposed and their specific affinities for certain diseases. We do not know the happenings in the brain of the healthy, the predisposed and the diseased. Therefore we are often unable to appreciate what the real cause is, and what it only seems to be.

The laws of conservation of energy and psychophysical parallelisms are applicable within the psychical sphere. The question is: Can science discover that application? Experimental psychology can explain many psychical phenomena. Self-observation combined with the observation of others is the best method of investigating psychical phenomena. We have made little progress during the last decades in the scientific exploration of psychological disturbances. There has been a dearth of psychology, of the knowledge of psychical casuistics. Pathological anatomy has become the only hope of the future, according to the general belief; and this the writer endeavors to combat by his explanations.

2. *On a Peculiar Katatonic Symptom-Picture Removed by an Operation.*—Bonhoeffer declares that so-called katatonic symptoms are not pathognomonic of a special disease. Negativism, flexibilitas cerea, verbigeration,

rhythmical movements, and stereotpy are observed in diseases altogether unlike. Even if such symptoms are permanent, one has to be cautious in making the diagnosis "katatonic dementia." The operated case reported by the writer shows that on the basis of traumatic hystero-epilepsy symptom groups occur, very much resembling the later stages of katatonia.

3. *Ophthalmoplegia Interna Traumatica.*—Ernst Schultze reports a case in which an isolated paralysis of the ciliary muscle and sphincter iridis existed. Ophthalmoplegia interna is mostly found with tabes and paralysis caused by lues. But in this case it was most probably the result of a hemorrhage in the region of the oculomotor nucleus.

4. *The Trend of Investigation in the "Psychologic Studies."*—(To be continued).

(1903, No. 2, February 15.)

1. On the Psychophysiology of Negativism. RAGNAR VOGT.
2. Critical Remarks on H. Schmaus' "Lectures on the Pathological Anatomy of the Spinal Cord." F. NISSL.
3. The Trend of Investigation in the "Psychologic Studies." W. WEYGANDT.
4. Recent Studies on Alcoholism. HOPPE.

1. *On the Psychophysiology of Negativism.*—According to Vogt negativism can be explained like the other katatonic symptoms by the supposition of a higher "persevering" power of the cortex. The perseverance of one motor impulse diminishes the susceptibility to other movements. In many cases the patients try to make a requested movement, but during this trial contrary impulses arise. If you ask a katatonic patient to shut his eyes, sometimes opening and closing movements vie with one another, until finally the eyes are suddenly closed.

Vogt believes that with every movement of a group of muscles the antagonists also become innervated. This condition of innervation of the muscles, and the intensified "perseverance" of the psychomotor phenomena in katatonics, explain negativism sufficiently. If now katatonic patients find often that they have great difficulty in making the required movements, they think such requirements a vexation and they become morose, thinking it perhaps unnecessary to try to fulfil the request. Thus negativism appears frequently to be wilful perversity.

2. *Critical Remarks on H. Schmaus' "Lectures on the Pathological Anatomy of the Spinal Cord."*—Nissl says that these lectures contain all that we know at present about pathological changes of the medulla spinalis. He thinks it important that the author is a pathological anatomist, while before him neurologists and psychiaters have described the pathological anatomy of the nerve centers. This separation of one branch leads to one-sided opinions about it. Schmaus has tried to describe the anatomical changes in the spinal cord more correctly than it has been done before, from the anatomical standpoint. Nissl objects to the co-operation of a neurologist, to whom Schmaus committed the clinical part of his lectures. Then he alleges that Schmaus is in error regarding the lymphatic spaces. Nissl claims to have proven by experiments that the adventitious sheaths are the only true lymphatic spaces, and that the membrana adventitia of the vessels forms a biological margin between the ecto- and meso-dermic structures of the nervous system.

3. *The Trend of Investigation of "Psychologic Studies."*—Weygandt gives a full review or critique of the first volume of reports issued from the laboratories under the direction of Emil Kraepelin of Heidelberg.

4. *Recent Studies on Alcoholism.*—To be continued.

(1903, No. 3, March 15.)

1. On Hydrotherapy in Psychoses. W. ALTER (Leubus).
2. The Trend of Investigation in the "Psychologic Studies." W. WEYGANDT (Concluded).

3. Recent Studies on Alcoholism. Hoppe (Konigsberg).

1. *"On the Hydrotherapy of Psychoses."*—The author considers hydrotherapy to be the best of all sedative measures. He has tried it in 79 per cent of his patients, not only on cases of furious excitement and grave acute insanity, but also on deeply depressed, hysterical and demented patients. He employed cure-baths of 1 to 6 hours' duration; prolonged baths, not less than twelve hours, and wet packs continued from one and one-half to twelve hours. The cure baths were given to sleepless patients, and to paranoiacs having outbreaks or exacerbations. The emotional manifestations of hebephrenic and katatonic patients could be largely controlled by cure-baths. For hysterics the wet packs surpassed the prolonged baths. In the remissions of paresis prolonged baths of gradually lowered temperature seemed to lengthen the period of remission.

Baths lasting more than twelve hours have been an absolutely effective remedy in all states of grave excitement; also in subduing hallucinations in dementia præcox, the prolonged baths have been very useful. The author did not in any case abandon the prolonged bath on account of a negative result. Repeatedly he continued it for very long periods. Occasionally it was continued six, eight, fourteen and twenty-three days. The author mentions a case of puerperal "exhaustion psychosis," in which a cure resulted from a bath of seventy-two hours. In mild hysterical cases and in convalescent patients who cannot sleep, Alter recommends wet packs: arteriosclerosis and heart disease do not contraindicate the measure. Hydrotherapy requires the strictest individualization to secure the benefits of it; but properly used, it permits a great reduction in the use of calmative drugs. Alter nevertheless gives opium to melancholics in increasing doses, supporting the medication by systematic wet packs which always hasten the effect of the drug. Generally all psychoses had a milder, more subacute course under the new treatment.

3. *Studies on Alcoholism.*—To be continued.

(1903, No. 4, April 15.)

1. On Paralyses after the Employment of Creasote Phosphate. L. Loewenfeld.

The writer believes that his observations upon this condition are unique. Creosote phosphate, under the name "Phosphot (e)," is put on the market by a manufacturing chemist as a remedy against tuberculosis. A colleague of Loewenfeld's tried the preparation in three cases of pulmonary tuberculosis in doses of one gram injected into the gluteal mass. Good results were observed, as regards the lung affection, after 15 or 16 doses; but in each case paralytic manifestations in the upper and lower extremities appeared. The manufacturers of the drug say that others have reported similar results. Loewenfeld gives a full description of two cases of this paralysis. The lower leg- and foot-muscles are first affected; the knee-jerk remains; sensory symptoms are slight; bladder and bowel are not affected. In one of the cases atrophy was present for some time in the peroneal region. The writer gives reasons for thinking that the paralysis is not altogether peripheral, and concludes that this, like most toxic paralyses, is a neurone disease, the ventral gray horns and the peripheral motor nerves being affected simultaneously. The phosphoric acid part of the compound surely cannot be the cause of this palsy; and the creasote in it is only half of what is contained in "creasotal," so that Loewenfeld is at a loss to account for this untoward effect of creasote phosphate.

Pickett (Philadelphia).

### JOURNAL OF MENTAL SCIENCE

(Vol. 49, 1903, No. 1, January.)

1. Some Features in the Intimate Structure of the Human Cerebral Cortex. JOHN TURNER.
2. Insanity in Imbeciles. A. F. TREDGOLD.
3. The Training of Nurses in Institutions for the Insane. BEDFORD PIERCE.
4. Case Taking in Large Asylums. D. F. RAMBAUT.
5. Treatment of Phthisis in Asylums by Urea and its Salts. J. L. BUSKIN.
6. The Alkalinity of the Blood in Mental Disease. ROBERT PUGH.
7. The Abnormalities of the Palate as Stigmata of Degeneracy. E. H. HARRISSON.
8. Insanity from Hasheesh. JOHN WARNOCK.

1. *Structure of the Human Cortex.*—These new features described by Dr. Turner are (1) A beaded net-work which involves pyramidal cells of the cortex cerebri and which have not, hitherto, been observed in the human brain, but only around the nerve cells of some of the lower animals (guinea pigs and rabbits) when subjected to the influence of methylene blue injected into the tissues during life; and (2) an intercellular plexus of extremely fine fibrils which has, he believes, never before been actually demonstrated in any brain, human or otherwise.

The technic employed by the author is a modified methylene blue method, as described in the autumn number of Brain. 1900. The pericellular network which he has first described for human brain consists in man of fine dark fibrils, on which, at varying distances, are small dark beads, or sometimes rings, which, as a rule, are the nodal points of the meshes. The size of the mesh and the coarseness of its fibrils also differ, so that sometimes one meets with a big-meshed net, having relatively few beads and with very delicate fibrils, at others the beads are larger, very closely clustered together or even partially coalescing and the fibrils much coarser. So far as he has yet been able to determine, this network is met with only within the cells of the pyramidal type. It could be seen over these in all the layers where they are met with except the skin layer and probably the innermost or lower of the spindle-cells. This network he believes to be of nerve structure, as it is found along the dendrites, and this is evidence, he believes, that these parts themselves are concerned in the conduction of nervous impulses. On the question of thorns the author believed these to be artefacts. The paper is well illustrated and is one of much suggestive interest, particularly as the author takes up a number of related physiological questions.

2. *Insanity in Imbeciles.*—Insanity is rarely met with in the more pronounced grades of amentia. In the middle grades of imbecility, however, such a complication is far from being infrequent, and out of over 200 imbeciles whom the author has examined in the asylums of the London County Council, more than one-half at one time or another had been insane. The subject has not been discussed at all in England, and it deserves more attention. It is found, he thinks, that those imbeciles who subsequently became insane have for some years before the actual outbreak been prone to sudden fits or irritability, bad temper, moroseness or sulkiness often accompanied by acts of violence; or that they have been in the habit of wandering away from home, or that they have evinced a restless disposition. This mental instability appears to the author to be by far the most important factor in the causation of insanity in these patients. Uncontrollable ideation, with resultant delusions, uncontrolled emotional action are two very characteristic features in these imbeciles. They have emotional storms which are very common.

As for clinical types of insanity, the author says these are mania or melancholia, mania being the most common form occurring in about 55 per cent of all cases: melancholia (probably the older definition of melancholia here being meant) in 40 to 45 per cent. Delusional insanity is very rare and general paresis occurred in only two or three per cent.

3. *The Training of Nurses.*—This is a short paper on the general topic of administration that contains very little that can be abstracted. The author in general praises the methods that are practised in our own institutions, and speaks of them as being adaptations of general hospital methods to asylum life.

4. *Case Taking.*—A short note on how to keep case books.

5. *Treatment of Phthisis by Urea.*—This is a short paper on the action of urea in the treatment of tuberculosis, in addition to some remarks about infection and disinfection in asylums. The physiological chemistry quoted in the paper is very fragmentary and belongs to an earlier generation. As to the exact value of urea in tuberculosis the latest researches do not bear out Harper's extravagant claims.

6. *Alkalinity of the Blood in Mental Disease.*—R. Pugh pursues his method for determination of the alkalinity of the blood as already spoken of in our abstract of his article on alkalinity of the blood in epilepsy in Brain already presented, q. v. He summarizes his work on epilepsy in the present paper and discusses the blood conditions in general paresis, mania and in melancholia. His conclusions are as follows: (1) The alkalinity of the blood is physiological in chronic mania, melancholia and dementia. (2) It is lowered in cases of mania, during the period of excitement. (3) It undergoes marked variations in epilepsy, e. g.: a. It is below normal during the inter-paroxysmal period; b. it undergoes a sudden and pronounced fall immediately prior to the onset of the fit; c. it undergoes a further diminution after the fit is over. This after-diminution depends upon the length of time, the severity of the muscular spasms, and the degree of the alkalinity in the inter-paroxysmal period; d. there is a gradual return of the blood to its normal alkalinity, which takes place in five to six hours; e. there is a relationship between the degree of the alkalinity and the onset of fits, e. g., the higher the alkalinity the less liable is the patient to have a fit; f. it is impossible to elevate and maintain the alkalinity within physiological limits for any appreciable length of time by the administration of drugs. (4) It undergoes a diminution in dementia paralytica. This diminution is constant and well marked, and is probably due to the products of degeneration in the circulation. The variations in the diminution met with depend upon the type, progress and duration of the disease.

7. *Abnormalities of the Palate.*—The author first gives a summary of the various points of view of others, and the relationship between types of palate and mental qualities and describes twelve different kinds of palates and comes to the amazing inference: The palate of insane heredity is essentially a palate which is shallow or, at any rate, of the average depth in front, whatever its other characteristics may be. His general conclusions are summed up as follows: (1) Abnormalities of the palate are common in the insane; (2) these abnormalities may be roughly classified into two large groups, of which the former contains the palate of the hereditary psychopath and the latter the palate of the general degenerate; (3) the former palate is variable in its general type, but as a whole is shallow, or, at any rate, of the average depth in front; (4) the latter palate is also variable in its general type, but is in the main characterized by an increased depth at the level of first bicuspids.

The general criticism that may be offered is that the author makes generalizations from the few cases studied rather than from a general survey of the whole field.

8. *Insanity from Hasheesh.*—The author's experience in Egypt as di-

rector of the Egyptian Hospital for the Insane at Cairo puts him in a position to speak authoritatively on this interesting topic. Large quantities of cannabis are used by the inhabitants of the Egyptian towns, where the drug is mostly consumed by smoking in the gozeh and in cigarettes. Hasheesh users usually degenerate morally, and for this reason, if for no other, the habit is disapproved of in Egypt. Insanity from hasheesh belongs to the toxic group and in many regards is similar to that caused by opium, alcohol, cocaine, etc. The author compares the insanity from hasheesh along lines known for alcoholic intoxication. Thus he describes (1) Temporary intoxication, in which the smoker becomes dull and drowsy, feels pleasantly exalted and is at peace with the world. This is the common state to be observed among habitués of hasheesh cafes. There is very frequently a staggering gait, and if the hasheesh smoker is annoyed or interfered with he is liable to become excited, irritated and loses control. The author contrasts hasheesh, opium and alcohol by saying that the mental pose of the hasheesh smoker is more subjective than alcohol and less so than that of the opium user. The alcoholic is the most objective and demonstrative of the three. (2) Delirium from hasheesh, which is usually accompanied by hallucinations of sight, hearing, taste and smell. There is great exaltation, much restlessness and sleeplessness with motor phenomena of staggering, although there is no tremor or ataxia. Physical excitation and gastro-intestinal disorders do not occur. (3) Mania varies in degree of acuteness from a mild, short attack of excitement to a long, furious mania, ending in death. (4) Chronic mania may result. (5) Chronic dementia usually is a final stage of the preceding forms. (6) Cannabinomania is employed by the author to describe the condition of many hasheesh users between the attacks of the other forms. The user is usually a good-for-nothing, lazy fellow, who lives by begging and stealing, and pesters relatives for money to buy hasheesh.

The author gives some statistics bearing on the amount used, and a number of interesting and important legal suggestions. He believes that hasheesh is a much more dangerous drug in Egypt than India.

(Vol. 49, 1903, No. 2, April.)

1. Bacteriological and Clinical Observations on the Blood of Cases Suffering from Acute Continuous Mania. L. C. Bruce.
2. The Case of an Unrecognized Degenerate Punished by the Law. E. Goodall.
3. Nomenclature of Mental Diseases. A. R. Urkuhart.
4. The Care and Treatment of Persons of Unsound Mind in Private Houses and Nursing Homes. E. W. White.
5. Lunacy and the Law. T. O. Wood.
6. Note on a New Case Book Form. W. R. Dawson.
7. Notes on Hallucinations, II. Conolly Norman.

1. *The Blood of Acute Continuous Mania.*—L. C. Bruce of Murthly contributes a series of observations on the blood in acute mania. A few years ago he made some observations on the blood in insanity and was unable to find any micro-organisms and he devised a new method of making aseptic necrotic areas subcutaneously and then subsequently studying this area to ascertain the bacteria found therein. Acting on this theory he took a case of acute mania, an adult woman, and with antiseptic precautions injected into the soft tissues of the flank two c.c. of turpentine. An abscess formed and on the third day after the injection he aspirated some fluid and obtained a small culture of a small diplobacillus. Since this time he has made 24 similar observations and has observed the diplobacillus in seven instances. It has been found only in

cases of acute continuous mania, although other forms were studied. The details of the cultural characters are given. In a further portion of his paper he gives the results of the study of the leucocytes in fifty cases of acute insanity. Adopting 6,000 to 10,000 leucocytes per cubic mm. as his normal, he has studied leucocytosis in those who became chronic and those experimented on as previously reported and recovered, and in those in whom abscess was made in a chronic case which did not recover. In recent cases he finds that there is a high leucocytosis, thus bearing out the later contention that acute manias are always of infectious origin. This leucocytosis is mostly in the polymorphonuclear cells. In the second class of cases the leucocytosis tends to remain between 12 or 60 and 16,000, but the polymorphonuclear cells are rarely over 50 per cent. In his third group of cases leucocytosis was high, but gradually sank and recovery took place, mental improvement, the author declaring to be in proportion to the leucocytosis. His general conclusions are as follows: (1) If these blood observations are correct, they practically prove that acute continuous mania is an acute infective condition, and that when recovery takes place a condition of immunity is established. (2) They prove that, although the patient apparently recovers, the disease remains latent; hence the persistent leucocytosis, a point which might be of great importance in life-insurance examinations; (3) an examination of the blood is a valuable aid to prognosis, as for instance: If a case of mania has lasted for a month and remains maniacal and sleepless. The blood examination gives a leucocytosis of 14,000 per c.mm. of blood, with a percentage of 60 or below 60 of the multinucleated cells. The chances of an immediate or early recovery are poor. On the other hand, if the blood examination gives a leucocytosis of 18,000 or 20,000, with the multinucleated cells in a percentage of 70 or above 70, the prognosis is good. It is well when examining the condition of the blood to aid prognosis, to examine the blood on at least two consecutive days.

2. *Degenerate Punished by the Law.*—The case is that of a man, thirty-five years of age, who showed, on transferral to an asylum from a prison to which he had been condemned for twelve months' hard labor, symptoms of congenital deficiency with depressions, visual and aural hallucinations and agitation. The patient's ancestry was bad, and he had been in jail nine times previously. Anthropological measurements are also given in the paper. The author's conclusion is that the type of punishment meted, i. e., that of short-term imprisonments and discharge for this class of cases is useless and injurious to the individual and unjust to society. The prison is not a proper place for this type of criminal. He is mentally diseased and should be shut up for life in an asylum or reformatory.

3. *Nomenclature of Mental Disease.* The author gives some suggestions on the classification of the insane in which there is very little new and nothing striking or noteworthy.

4. *Care of Insane in Private Houses and Homes.*—The paper the author considers the natural outcome of Sir William Gowers' recent address on the care of the insane. It takes up the advantages of certified single care, and that they are privacy, domesticity, visits of friends, avoidance of the stigma of treatment in a lunatic asylum; whereas the disadvantages are the absence of skilled medical treatment, unskilled nursing, monotony, insufficient moral control, interference of friends, limited supervision, want of tact and business capacity on the part of the custodian. These are the advantages or disadvantages in the care and treatment of the duly certified person of unsound mind in private houses. As to the taking charge of a person of unsound mind not under service in a private house or nursing home the author's advantages and disadvantages are much the same, the want of official supervision being perhaps the most

serious of all the disadvantages. There are a number of useful suggestions in the paper, but they do not have very ready usage here.

5. *Lunacy and the Law.*—T. O. Wood discusses several features in the English Lunacy Law, which are of secondary interest to those practicing under different regulations.

6. *Note on a New Case Book Form.*—This is a short note on the method of keeping clinical histories and has no particular points one way or another.

7. *Hallucinations.*—The author here gives the histories of thirteen patients suffering from different forms of hallucinations. The paper is too detailed to admit of ready abstraction, but it contains many interesting suggestions, bearing more particularly upon the theory of psycho-motor verbal hallucinations, whereas he does not discuss any of this type. Those that are given show interesting connections with the forms described by Seglas.

8. *Clinical Notes and Cases.*—This number also contains Notes on some Pathological Cases, by M. J. Nolan, the swallowing of foreign bodies, such as nails, bristles, etc., by dements; a case of suppurative cholangitis; melancholia with universal acute eczema; senile melancholia associated with aneurisms of the aorta and rupture, and also a few Cases of Abdominal Surgery in the Insane, by Robert Jones; A Case of Hebephrenia, by W. R. Dawson; a Case of Thoracic Aneurism in a Secondary Dement, Simulating Mediastinal Growth, by Robt. Pugh; and Status Epilepticus Complicated with Scarlet Fever, by G. W. Greene. JELLIFFE.

## BRAIN

(Vol. 25, 1902, Winter, No. 4.)

1. Some Observations on the Primary Degeneration of the Motor Tract. F. W. MOTT and A. F. TREDGOLD.
2. On the Pallio-tectal or Cortico-mesencephalic System of Fibers. CHARLES E. BEEVOR and SIR VICTOR HORSLEY.
3. A Case of Arrested Development of the Cerebellum and its Peduncles; with Spina-Bifida and Other Developmental Peculiarities in the Cord. W. B. WARRINGTON and K. MONSARRAT.
4. Toxic Degeneration of the Lower Neurones, Simulating Periphery Neuritis. STANLEY BARNES.
5. On Certain Blood Changes in Idiopathic Epilepsy. ROBERT PUGH.

1. *Primary Degeneration of the Motor Tract.* The authors describe the clinical course and microscopical appearances in four cases: two amyotrophic lateral sclerosis, one of progressive muscular atrophy and one of chronic rheumatoid arthritis with muscular atrophy. They say that degeneration of the motor tract is the anatomical lesion present in severe diseases having distinct clinical features. Thus it occurs in Friedreich's disease, in combined lateral and posterior sclerosis and in general paralysis, but in these other neurone systems are also affected. Degeneration may also result from local lesions as in the anterior poliomyelitis, polioencephalitis, embolus, etc. And lastly, cases exist in which not a degeneration but an arrest of the development of the motor tract occurs, apparently due to simple agenesis. It is with the primary degenerations, however, that occur in amyotrophic lateral sclerosis, progressive muscular atrophy, bulbar paralysis or primary spastic paraplegia that the paper deals.

Speaking of the nature of the morbid processes they believe that the changes in these cases are those of a sub-acute or chronic degeneration of a portion or the whole of the motor tract, that is of the upper segment alone, of the lower alone, or of both segments together. The cases they describe demonstrate that the change is a slow attack of individual neurones. The cells become shrunken and irregular in outline, the Nissl bodies gradually disappear, their space is taken by a large collection of yellow pigment, the nucleus becomes smaller and its outline displaced. Later it is displaced to the periphery of the cell and finally disappears. There is true degeneration in the nerve fibers and in the end organs. Neuroglia gradually takes the place of the atrophied nerve fibers causing a secondary sclerosis. The authors believe that this progressive degeneration is due to the fact that the neurone has reached the stage at which its inherited vitality is exhausted and that this presents a probable indication of presenile decay, but they present no evidence one way or another.

On the question of the pathological relations between the upper and lower motor segments they say that while there can be no doubt of the existence of trophic action between the upper and lower parts of the efferent tract, in some cases the presence of degeneration in one portion resulting from degeneration in the other seems to be rather the exception than the rule, and they consider that it is now well established that disease may involve either segment separately or both together, and if each segment of the motor tract is specially susceptible to the influence of particular poisons it seems legitimate to conclude that there may be different hereditary tendencies in the two tracts, in some persons there may be a premature tendency to decay in the upper portion. in others in the lower portion, and in still others throughout the entire motor system. Other neuronic involvement was present in some of the cases. These were considered secondary by the authors and were discussed under two heads, association fibers and efferent fibers.

2. *Pallio-tectal System of Fibers.*—An investigation of fibers composing the crus cerebri in a number of animals, 20 cats, 11 monkeys, 1 dog and 2 badgers. Local lesions in the cortex had been made by Horsley. While tracing the degerations which resulted from such lesions the authors especially followed out the connections which have for some years been known to exist between the cortex and the mid-brain. The Bush modifications of the Marchi method were used and serial sections studied. The results obtained are very detailed and the original should be consulted, but the general statement may here be made that as the research was founded chiefly on observations on the cat, representing the carnivora, and the macaque monkey, representing the primates, the results are to be understood as applicable only to the higher of these animals in which there is a well-developed non-excitable area (frontal) in front of the so-called motor or excitable cortex. The general scheme of the pallio-tectal system is stated by the authors to be as follows: From the frontal cortex no fibers can be traced to the mesencephalon; from the temporo-sphenoidal cortex very few fibers; while from the occipital lobe a fair number, and from the excitable cortex a large number of fibers can be followed to the corpora quadrigemina and mesencephalon. The course taken by the different group of fibers is then traced by the authors. We can not give the details in this place, but refer the reader to the original.

3. *Arrested Development in the Cerebellum.*—The authors describe in full the clinical history of a case of restricted development of the cerebellum, and then describe the macroscopical appearances of the brain and cord; the histology of the medulla, cerebellum, pons and membranes with the following results. A child, aged six weeks, presenting a lumbar spina bifida ruptured at or before birth, and now healed; hydrocephalus, paraplegia of both lower limbs. Death from intercurrent gastro-enteritis.

(1) In the brain. Arrested development of the cerebellum and of its efferent and afferent fibers, and of their nuclei of origin; of the restiform bodies, the olives, accessory olives, external arcuate fibers and nuclei arcuati, middle peduncles and transverse fibers of the pons, pontal nuclei, vestibulo-cerebellar tracts, superior peduncles, gray nuclei of cerebellum, red nuclei of tegmentum; possibly the cerebro-pontal tracts.

(2) In the cord. General attenuation of this structure throughout, division at the level of the fourth dorsal vertebra into two equal halves; marked enlargement of the central canal which divides with the division of the cord; the presence of various irregular cavities in the cord and disintegration of its central parts in the lower dorsal region; normal development of the gray matter with well-formed ganglion cells; scanty development of white matter, its place being taken by tissue of neuroglial structure.

(3) The coverings of the cord. The whole cord as far as the upper cervical area, embedded in a fine connective tissue with large vascular channels; the laminæ and spines of the fourth and fifth lumbar vertebræ deficient; an exostosis dividing the canal at the level of the fourth dorsal vertebra. In an appendix they give a complete bibliographical summary of cerebellar atrophies aranged on the basis of their proper pathogeny.

4. *Toxic Degeneration of the Lower Neurones.*—Dr. Barnes reports the histories of seven cases in which the clinical picture resembles in many respects that of peripheral neuritis and yet in other ways corresponds to the type of progressive muscular atrophy. The clinical histories are given very thoroughly, and his conclusions are very interesting. These may be summarized as follows: (1) There is a clinical type which is usually the sequel of acute specific fevers, which resembles the paralysis seen in multiple neuritis, but which is associated with great atrophy of the hand muscles. It usually begins about the second or third week after the febrile condition and involves the muscles from the periphery to the trunk to a varying extent. It may progress for a few days only, or for several months. Sensory signs are present. but slight in degree. Although the condition somewhat resembles progressive muscular atrophy, the prognosis and etiology of the two conditions are probably widely different from one another.

(2) After a certain stage, when once definite improvement has begun, relapses are not common, and there is a constant tendency to improvement. Even years after the subsidence of the acute condition considerable improvement may still take place, the small muscles of the hands being the last to recover. Contractures are rarely developed.

(3) Probably the condition is one of toxic degeneration of the lower neurones, the motor neurones being more particularly affected.

5. *Blood Changes in Idiopathic Epilepsy.* Dr. Robert Pugh of Claybury presents the results of the examination of forty cases of epilepsy in which differential counts of the leucocytes have been made, and the study of the alkalinity of the blood carried on. The author concludes that in epilepsy there are marked variations in the alkalinity of the blood, most of which manifest themselves by diminution of alkalinity. The average alkalinity during the (a) interparoxysmal period is lower than the average of the control cases. (b) There is a sudden pronounced fall immediately prior to the onset of the fit. (c) There is a certain diminution after the fit is over. Accounting for the interparoxysmal diminution the author suggests two hypotheses: (1) It may be explained by the gradual accumulation of toxines of an especial nature in the blood, and (2) that it may be the result of deficient metabolism, general metabolism being known to be below par in epilepsy. Accounting for the pronounced fall in the alkalinity immediately prior to the onset of the fit. The most likely origin of the acids the author claims are either (a) neuronic, (b) muscular. In the first case, the increased amount of acid being derived from the muscular and the glandular tissues. The author says that the exact rôle which the diminution in the alkalinity of the blood plays in the production of an epileptic attack is difficult to estimate. It is of utmost importance for any

cell in the body to have a proper adequate and pure supply of blood, and this applies especially to cerebral neurones, as they are extremely sensitive to a change in environment. The author believes that he has obtained good results in the treatment of epilepsy by maintaining the alkalinity of the blood within physiological limits, but unfortunately he says it is impossible to elevate and maintain the alkalinity by the administration of drugs.

With reference to the leucocytes, there is a distinct leucocytosis, particularly after the attack. The author's general conclusions are as follows: (1) The alkalinity of the blood in the interparoxysmal period is lower than the average of the control cases; (2) the diminution is gradual and progressive and is more marked in those cases suffering from gastric catarrh and constipation; (3) there is a marked sudden and pronounced fall immediately prior to the onset of the fit; (4) there is a further fall in the alkalinity after the fit is over; this diminution is seen from three to ten minutes after the attack; (5) this after-diminution depends upon the duration and severity of the muscular twitching, and upon the degree of the alkalinity in the interparoxysmal period; (6) there is a gradual return of the blood to its normal alkalinity, which takes place in five to six hours, the rise being more marked in the first hour.

Jelliffe.

## LE NEVRAXE

(Vol. IV. Fasc. 3. 1903.)

8. On the Form and Development of the Protoplasmic Prolongations of the Spinal Cells in the Higher Vertebrates. T. Geier.
9. Researches on the Central Acoustic Tracts: The Bulbo Mesencephalic Acoustic Tracts. Van Gehuchten.
10. The Inhibitory Fibers of the Heart belong to the Pneumogastric Nerve and not the Spinal Nerve. Van Gehuchten.

8. *Form and Development of Protoplasmic Prolongations.*—Dr. Geier has been studying the dendrites of the spinal cells of the horn fibers, using kittens and rabbits for investigations. Golgi's method was used. He finds that the dendrites of the anterior horns are smooth in contour and differ from the posterior horns which have an irregular contour. The spinal dendrites extend in a straight line, while those in the posterior horn are irregular and sinuous. The dendrites in the cells of the anterior horns have few and insignificant collateral appendices while those of the posterior horns are quite complex. There are many variations in the dendrites of the new-born animal as contrasted with those of the adult, for full details of which the reader is referred to the original.

9. *The Upper Auditory Tract.*—Van Gehuchten here presents results of an extended series of experiments performed on rabbits and studied by the Marchi method. Five series of experiments are given which lead Van Gehuchten to different conclusions than those heretofore held. In the first group of cases he tore the seventh nerve out through the stylomastoid rim. In this way the ventral fibers of the trapezoid body were involved. Serial sections showed that degenerated fibers were found passing into the trapezoid body vertically above and below the actual level of the lesions. In the trapezoid these fibers could be traced to the opposite side; they turn upwards, those ventrad passing in front of and those dorsad behind the trapezoid nucleus. These two degenerated bundles so united to form a compact strand of fibers which lay in contact with the lateral aspect of the inferior nucleus of the lateral lemniscus. Tracing this strand of fibers upward it was found to turn outward and then backwards finally entering and forming the more superficial part of the lat-

eral fillet. From this place onward the fibers could be traced to the posterior quadrigemina where they terminated close to the superior nucleus of the lateral lemniscus. In the second series of cases, similar degenerations were found. In a third series a lesion was made by cutting downward and outward through the floor of the fourth ventricle, dividing both the dorsad and ventrad fibers of the corpus trapezoid. Similar conclusions as those already outlined were reached by this method of investigation. The fourth series of experiments was made to ascertain the cells of origin of the two strands of the corpus trapezoid tract. If a lesion was restricted entirely to the accessory nucleus of the eighth nerve degenerations corresponding to those just described resulted. On the question of the relation of the medullary striæ in the secondary auditory path the author found that attempts to cut these on the floor of the fourth ventricle were unsuccessful but they were found destroyed in an experimented animal in which the seventh nerve had been torn out and had ruptured the acoustic striæ. Degenerated fibers can be followed from the point of injury to their raphe where they crossed dorsad to the upper fibers and the trapezoid body until they became posterior to the olivary nuclei. They then turned upwards, lying behind the dorsal trapezoid tract and internal and ventrad to the motor fifth nucleus. They entered the lateral lemniscus at the upper border of the upper cerebral peduncle, being mingled with the fibers of the dorsal strand of the corpus trapezoid. They finally ended in the nucleus of the posterior quadrigemina itself, thus tracing the fibers of the secondary auditory path higher than those previously described. The author was unable to verify the opinion of Held that neither the dorsal nor ventrical auditory paths could be traced to the inferior corpora quadrigemina or cortex. Thus the author holds that the central auditory path is in reality a bulbo mesencephalic rather than a bulbo-cortical one. The illustrations are numerous, there being over 62 illustrations accompanying this extremely interesting and suggestive article.

10. *Inhibitory Fibers of the Heart.*—Van Gehuchten controverts the old idea that the inhibitory fibers of the heart arise in the spinal accessory and pass to join the vagus by its internal branch.

He concludes that (1) the inhibitory fibers of the heart do not come from the spinal accesory nerve but are derived from the vagus itself. (2) His investigations on the innervation of the larynx (see abstract of last number) go to prove that all of the bulbar fibers of the spinal accessory are distributed to the muscles of the larnyx; the eleventh nerve has an exclusive medullary origin and (4) the vagus gives off a superior and inferior system of fibers exclusively motor, including motor fibers destined to all the muscles of the larynx exclusive of the crico-thyroid, which is supplied by the inferior laryngeal nerve and a secondary series of inhibitory fibers to the heart. JELLIFFE.

### REVUE DE PSYCHIATRIE ET DE PSYCHOLOGIE EXPERIMENTALE

(January, 1903.)

1. Tests in Psychopathology. ED. TOULOUSE and N. PIÉRON.
2. Experimental Researches on the Psychology of Memories. N. VASCHIDE and CL. VURPAS.
3. Study of Morbid Obsessions.

1. *Tests in Psychopathology.*—The aim of this article is to indicate the lines along which the development of psychopathological tests must proceed if they are to be more exact and comparable. In their introduction the authors critically review the work of Guicciardi and Ferrari and of Tamburini, Badaloni and Brugia. They then proceed to a brief statement

of their own methods illustrated by their application in the domain of memory. They believe in the general principle that the methods for the study of the abnormal mind should be the same as those used for the study of the normal, for the abnormal mind differs only from the normal in showing exaggeration of certain characters or defect of others—differences only of more or less. Applied to an experimental study of the memory of sensations their method would involve the use of a definite multiple of the previously determined minimum perceptible stimulus in the sensory field under consideration. In this way only can results comparable as between different subjects be obtained as no stimulus is felt the same by all. There is no absolute in psychology, only the relative. In the study of the memory of perceptions the optimum or quantity best perceived is used as a basis, while in the study of the memory of ideas sentences constructed of identical length, in the same style, containing the same number of ideas disposed in the same way are used, thus detaching as far as possible the idea from its setting. The article closes with a few words on the calculation of errors.

2. *Psychology of Memories* (continued).

3. *Morbid Obsessions.*—The authors have studied for this article in all ninety-seven cases. They have studied only those cases in which the obsession was the essential psychopathic symptom, not differentiating for their purposes the phobias from the morbid obsessions. The results of their studies are as follows:

Of the 97 cases examined 76 were men and 21 women. The occupations were distributed as follows—8 clergymen, 11 merchants, 11 office employees (all men), 14 domestics (all women), 17 artisans, and 29 persons in different intellectual professions. The greater part of the obsessed were thus from intellectual pursuits; 92 per cent of all cases showed hereditary tendencies. Of 94 cases 7 only showed no afflicted relatives, 38 showed 1 or 2, 30 cases had 3 to 5, and 19 cases 6 or more. Thus in the great majority of cases at least 3 members of the family had been afflicted. Of 88 cases 7 showed no heredity, in 32 heredity existed on one side only, and in 49 on both sides. One-third of all cases showed a homogeneous heredity. A study of the mental life of these cases shows them to have been possessed of a peculiar character from early life, marked by indecision, irritability, desire for solitude, painful and disagreeable emotions, egotism and often hypochondriacal ideas. In 77 of the cases the phenomena manifested themselves at twenty years of age, in 11 cases after twenty years. The development was usually progressive and by insensible degrees. This progress, however, may be interrupted by exacerbations, especially as a result of influenza, menstrual difficulties, puberty, etc. Occasionally as an accompaniment of the obsession is found epileptoid, hysteriform, vertiginous or syncopal attacks, and sometimes complicating this condition sexual perversion, sexual excitability (4 cases), masturbation (42 cases), or other sexual anomalies (28 cases). Occasionally these cases dement, and then arises the question of differential diagnosis between pure morbid obsessions and dementia præcox with obsessions.

(February, 1903.)

1. The Mental State in Cerebral Tumors. A. VIGOUROUX.
2. Experimental Researches on the Psychology of Memories. N. VASCHIDE and CL. VURPAS.

1. *Mental State in Cerebral Tumors.*—The author deals with these three points in this article; first: have these psychic troubles characters which point to the presence of a tumor? second: are they of such a nature as to indicate its location? third: can they be classified? After a careful review of the opinions of writers on this subject the authors answer these queries negatively. Psychic symptoms may or may not accompany a tumor in a given location, and their nature is independent of the nature of the tumor as such and may be the result of accidental conditions, such as inter-

ference of circulation, meningitis, the impregnation of the brain by toxic products of disassimilation, surrounding inflammation and many other causes. Finally the author concludes, with Ball, that when a true psychosis is found associated with a cerebral tumor, there is every reason to believe that the association is a simple coincidence.

2. *Psychology of Memories* (concluded).—An experimental study confined solely to the domain of psychology and having little interest from the standpoint of psychiatry. WM. A. WHITE (Binghamton, N. Y.)

## MISCELLANY

HYPNOTICS AND THEIR DANGERS. Dr. Nestor Tirard (Lancet, April 11, 1903).

In a treatise on some dangers of hypnotics the author draws the following conclusions: He says that he is convinced that in the employment and in the selection of a hypnotic sleeplessness is perhaps the least important factor to be considered, although it may be the sole symptom of which complaint is made. The cause must be ascertained, together with any indications of idiosyncrasy. The co-existence of other diseases must influence the selection of the hypnotic, and even when, for the individual case, a suitable hypnotic has been found, risks of repeating moderate doses must be borne in mind. Generally the dose has to be gradually increased, especially when the cause has not been ascertained or satisfactorily dealt with, but with some drugs, e.g., sulphonal, trional and tetronal, the danger appears to lie in the total quantity taken, even though each dose may have been small. But perhaps the greatest danger of all is incurred when the patient is given a prescription for a hypnotic, and this danger has certain analogies with the medicinal employment of alcohol. Many of us believe that at the height of fever or in conditions of collapse, alcohol is often necessary, but it is never recommended to our patients for indefinite use, still less for increasing doses. Armed with a prescription for a hypnotic patients commonly go on taking the drug long after the relief of the special circumstance for which it was prescribed, and the prescription may be used to facilitate the acquisition of large quantities of a dangerous drug obtained, perhaps, at several establishments in small amounts. Notwithstanding the relief which many have derived from the use of hypnotics, he is in favor of any step which tends to prevent their administration, except by the medical man or under his immediate directions. He has seen so much of the dangers of hypnotics that he would prohibit their sale to the public or would allow them only to be dispensed to a freshly-dated prescription, which should be retained by the druggist or returned to the prescriber. JELLIFFE.

NERVE SUTURE AND NERVE DEGENERATION. P. B. Henriksen (Lancet, April 11, 1903).

In a study on the subject of nerve suture and nerve degeneration, the author says, considering the question of whether a degenerated nerve can be supposed to be to some extent conducting: This question may be solved in two ways—either by proving conducting elements in the degenerated nerve or by proving effects of the nerve function during the degeneration period. According to what has formerly been mentioned, the new fibers must be supposed to be formed of elements in the peripheral part of the nerve. What are these elements, and at what time can they be shown as continuing courses fit for conducting a nerve impulse? When a nerve is divided the weight of a corresponding muscle will diminish rapidly. By weighing the muscle twelve days after dividing one peroneal nerve of a rabbit it was found that the muscle corresponding to the divided nerve weighed 4.77 grams, while the muscle on the other side weighed 6.98

grams—a difference of 33 per cent. When the nerve is united again motility comes earlier than the electrical reaction. In several of the experiments the difference seemed to be considerable, as the animals could use the operated limb as well as the other limb long before electrical reaction was observed. If the muscles have thus resumed their activity, and in consequence of a return of nerve conduction, one may suppose that this also may be shown by changes in the microscopical appearance of the muscles or in the cessation of atrophy or by increasing weight. To examine these circumstances, the third series of experiments was made in the following way: The peroneal nerve was divided on both sides. On the one side one of the ends was pulled out through the slit in the fascia and fixed subcutaneously, while the slit for the rest was closed with catgut. In this way the nerve was prevented from uniting. On the other side the divided nerve was permitted to unite. The animals were killed after some days' interval, and both the united nerve and the central and peripheral ends of the non-united nerve were hardened for microscopical examination. The muscles supplied by the peroneal nerve were carefully dissected out as similarly as possible on both sides and were then immediately weighed. A piece of the muscles was cut out from corresponding places of the muscles on both sides and hardened in formalin for microscopical examination. JELLIFFE.

MASSAGE IN LOCOMOTOR ATAXIA. Dr. de Frumerie (Le Progrès Médical, Feb. 28, 1903).

In every manual treatment it is necessary first to determine whether the effect desired is stimulation or sedation. In tabes the author confines his massage to sedative measures, avoiding any brusque or energetic movements which so often lead to hyperesthesia. The suitable types of manipulation for this disease are superficial or deep effleurage, vibratory motion of the abdominal viscera, and prolonged compression, the last sometimes overcoming the lightning pains when energetic measures have had no effect. For paralysis of the bladder the organ should be emptied of its infected urine and one-third filled with boric acid solution; then the curved fingers are pressed down behind the pubes and gentle tremulous motion set up. The perineal region may be subjected to effleurage and continuous pressure. When there are contractures, one must avoid the contracted muscles, the flexors, and by every means possible try to strengthen their antagonists, the extensors. The flexors then should be slowly and cautiously stretched by passive motion. Where there are no special contractures, a general stretching or extension may be brought about as follows: The patient lies on a bed and places the feet against the chest of the operator; then, without bending his knees, he is seized by the hands and pulled forward, the operator at the same time giving him a tremulous movement. This affects the spinal cord, the sciatics, the ligamentous and muscular tissues of the back, and the brachial plexus, without fear of injury to the femoral neck, which is so fragile in certain tabetics. BASTEDO.

MYASTHENIA GRAVIS. Charles S. Myers (Journal of Pathology and Bacteriology, Sept., 1902).

Three signs may be described as pathognomonic of myasthenia gravis. They are (1) ready fatigue of certain or general voluntary movements either to a succession of tetanizing currents applied to the nerve or to volitional impulses descending from the brain. (2) The exacerbations and remissions shown in the course of the disease, and (3) the tendency to a fatal implication of the muscles innervated by the bulb. To these must be added other very important general signs mainly of a negative character. (4) There is no reaction of degeneration; the muscles react to a faradic current of normal intensity, or more often require one somewhat stronger than normal. (5) The muscles, as a rule, show neither atrophy nor fibrillar contraction. (6) Sensation and intelligence are unimpaired. (7) The reflexes may be normal, are often feeble, but perhaps most frequently are somewhat exaggerated. Clonus is absent. (8) Purely voluntary muscles are alone affected. The mechanisms of defecation and micturition are never impaired. (9) The myasthenia, although often unequally marked on the two sides, is almost invariably bilateral. Myasthenia gravis is distinguished from hysteria by absence of stigmata or disturbance of sensation; from clonic bulbar paralysis by the extraordinary remission and exacerbation of symptoms, the general absence of muscular atrophy, of fibrillary contractions and degenerative electrical variations. Of 22 cases on which autopsies were performed, 8 showed no pathological condition of the nervous system. In 3 cases minute aneurisms or vascular dilatations were noted; and in 2 the cerebral arteries appeared thickened. Eight cases examined by Nissl's method, in 3 slight chromatolysis was found. Hemorrhages in the central nervous system always recent and frequently numerous were found in 6 cases. The peripheral nerves and muscle fibers were examined several times, yielding varying changes in the muscles and rarely changes in the nerves. The writer does not believe that either the muscles or the higher parts of the brain are primarily affected. The disease probably arises from some autogenetic-toxine. The end plate is affected and with it the entire neurone, secondarily. W. B. Noyes.

The Diagnosis of Brain Abscesses. Herman H. Hoppe, M.D. (Journal American Medical Association, March 14, 1903).

Given a certain group of signs and symptoms which lead us to suspect the presence of a brain abscess, the first point is tracing the source of infection. Bergman says that without the Streptococcus pyogenes aureus there cannot be a brain abscess. But to find the external source of infection is difficult. Bergmann divides brain abscesses into four groups. (1) Traumatic abscess caused by an open scalp wound or a fractured skull. (2) Those abscesses resulting from a purulent inflammation of bone or bony cavities, (a) otitic abscess, (b) rhinogenetic abscesses, (c) abscesses due to caries of the bones of the skull. (3) Tuberculous abscesses. (4) Metastatic abscesses. The symptoms of brain abscess may be grouped as follows: (1) Symptoms due to deep-seated suppuration, (2) symptoms due to intracranial pressure, (3) focal symptoms. Fever and other signs of sepsis, chills, localized pain and tenderness, vomiting and, at times, distinct mental symptoms are important symptoms. Optic neuritis is more frequent than in brain tumor, but not so well developed. If it is absent the abscess is either very acute or small. It is most marked in abscess of the frontal lobe. Focal symptoms help much to make the diagnosis if the abscess is present in the psychomotor area, little if present in the occipital, temporal or frontal lobes. The closer the abscess to gray matter, and the greater the destruction of tissue, the more focal signs are developed. A large abscess of the centrum ovale may exist without focal signs.

W. B. Noyes.

# Book Reviews.

A TREATISE ON HUMAN ANATOMY IN ITS APPLICATION TO THE PRACTICE OF MEDICINE AND SURGERY. By JOHN B. DEAVER, Surgeon in Chief to the German Hospital, Philadelphia. In three volumes, illustrated by 499 plates nearly all drawn for this work from original dissections. Vol. III. Abdomen, Pelvic Cavity, Lymphatics of the Abdomen and Pelvis, Thorax and Lower Extremity. P. Blakiston's Son & Co., Philadelphia.

The arrival of the third volume of Deaver's Surgical Anatomy has been anxiously awaited by all possessors of the first two volumes, and their fondest anticipations have been fully realized upon their receipt of the final volume which has more than kept up the high standard already established by the first two.

The excellence of the plates from an artistic standpoint and their correctness as to anatomical detail, can hardly be surpassed. In the text the author has not gone into the minute anatomical description of the different organs, yet he has indicated most of the surgical procedures which may be required and has presented it in a clear and concise manner.

Volume III takes up the Abdomen, Thorax and Lower Extremity. The surface anatomy of the abdomen and thorax is beautifully depicted with tracings of the main vessels and contained organs, giving the anterior, posterior and lateral views; then follow descriptions of the superficial and deep fasciæ and the muscles. The anatomy of the inguinal canal is well brought out and is made perfectly clear by the numerous illustrations. The peritoneum, the reflections of which the student is apt to find a stumbling block, is made perfectly plain by means of four full page plates. The abdominal and thoracic organs are all described and beautifully depicted, particularly the heart and lungs. The lower extremity has its surface anatomy marked out and particular attention is given to the anatomy of Scarpa's triangle, the gluteal region and femoral hernia, also the popliteal space. The joints of the lower extremity are fully described, and the deformities commonly met with, as well as the surgical procedures indicated for their relief. Dislocations are also touched upon as well as excisions, the lines of incision for which are indicated not only for the joints themselves but for the bones of the foot. Fractures are lightly gone over, likewise amputations, ligation of arteries and nerve stretching.

The author has left little ground for criticism in this work, and the only one we could possibly make would be in reference to the female pelvic cavity. In a field where so much work is done at the present day, not only by the gynecologist but the general surgeon, we think a plate showing the relations of the ureter and uterine artery would be greatly appreciated, as the former structure is so frequently injured in doing a hysterectomy. Although the author advocates, as does Kelly, the passing of bougies into the ureters to mark their course, in reality this is seldom done, and frequently it is impossible on account of the tumor which necessitates the hysterectomy, distorting the bladder to such an extent as to render the passage of bougies exceedingly difficult, if not impossible.

The author tells us in his preface that as originally planned the work was to contain 200 illustrations. Vol. III contains 178 alone and the text has been constantly revised and much added, especially with reference to surgical procedure. He certainly deserves the congratulations of the medical profession, particularly the surgeons, for bringing to completion this work, which has cost him sixteen years of painstaking labor. Every practitioner doing any surgery will find this work of exceedingly great value, and the student of anatomy will find the illustrations a great aid to thorough comprehension of the subject. CHAS. F. ADAMS.

ORGANIC NERVOUS DISEASES. By M. ALLEN STARR, M.D., PH.D., LL.D. Illustrated with 275 engravings in the text and 26 plates in colors and monochrome. Lea Brothers & Co., New York and Philadelphia, 1903.

The author of this work is so well known, both at home and abroad, and his experience has been so extensive, that we may expect much from him in a book on nervous diseases. The statements made by him, as he himself says, are based in a large degree upon the pathological and clinical material accumulated during the past twenty years. A careful examination of the work shows that he has carried out his intention of utilizing his personal observations without ignoring neurological literature. The descriptions are clear, and the work, while it will be read with interest by the specialist in neurology, will be easily understood by the undergraduate student.

A short chapter on the structure of the nervous system is the introduction to several chapters on diseases of the nerves. These are followed by chapters on organic diseases of the spinal cord and of the brain. The neurone doctrine is taught.

In regard to trophic lesions, Starr says that the explanation of these changes is to be found in the interruption or perversion of sensory impressions and that "nature, lacking its accustomed guide to repair and misled by abnormal impressions, produces effects which are needless under the circumstances, or fails to produce those which ordinarily would be required." Physicians and nurses may read with prickings of conscience that bedsores may be entirely prevented if by extraneous care pressure and irritation are avoided, as in health would occur by change of position and care of the skin. This is not unlike a statement made by Strümpell years ago, and yet there are still physicians who are unwilling to explain bedsores entirely by mechanical causes. Starr has little belief in the origin of trophic disturbances in alteration of special trophic or of vasomotor nerves.

He has never seen a case of true ascending neuritis, although he has seen many cases of painful affection of the extremities above the level of the neuritis that were hysterical in nature. As regards those cases that are not hysterical, Starr's explanation is that "mild irritation long continued produces in a nerve center a hypersensitive state by what is known as a summation of impulses. In this state slight impressions are believed to be intense and local impressions become generalized and widely referred. This state may be induced by neuritis long continued, and has been mistaken for an ascending neuritis." Ascending neuritis has been regarded by other neurologists as an extremely rare affection, and yet some practitioners seem to make the diagnosis in ignorance of this fact.

Starr believes that neuralgia has, in every chronic case, some organic lesion, and that in acute cases some temporary malnutrition of the sensory fibers is present. It is hard to doubt the correctness of this statement, especially if one has made many clinical and pathological studies. Starr accepts this teaching so readily that he does not hesitate to place neuralgia among the organic nervous diseases. Aconitine is the best remedy, in his opinion, for tic douloureux, and he administers it in pill or tablet form, preferring the French pills of Chapoteaux. Each of these contains 1-500 grain, and the remedy must be used with sufficient frequency to produce constitutional effects. He begins with one pill every four hours and decreases the intervals each day one-half hour, until one pill every two hours is taken or symptoms are present, such as tingling of the tongue and fingers, a sense of general weakness, and feebleness of the pulse. He combines with the aconitine a fiftieth of a grain of strychnine during the first two days, and then 1-100 of a grain when the aconitine is being given every two hours. Sometimes he substitutes caffeine or sparteine for the strychnine. A cardiac stimulant should be used when large doses of aconitine are given, and sudden muscular efforts should be avoided. Other remedies are mentioned. As regards the strychnine treatment in massive doses. as recommended by Dana, Starr says he has seldom seen any benefit result from this treatment, but has seen severe strychnine poisoning ensue. We should like to have further opinion in regard to the aconitine treatment, because most cases of severe tic douloureux are treated successfully usually by operation.

In Starr's experience all syphilitic nervous lesions, whether central or peripheral, yield more promptly to the combined use of inunctions of mercury and large doses of iodide of potassium, than when either is employed alone. While he does not deny the possibility of multiple neuritis occurring as a result of syphilis, and even mentions two or three possible cases he regards this cause as very rare. Alcoholic neuritis, in his experience. as in that of Gowers and others, is relatively far more frequent among women than among men. It is important to emphasize the word *relatively*, as alcoholism is so much more common in males.

Starr does not attempt to distinguish sharply between chronic anterior poliomyelitis and progressive muscular atrophy of spinal origin; indeed, the latter is regarded by him as one of the types of the former. The sharp distinction made by some authors between these two forms of muscular atrophy has always seemed to the reviewer somewhat arbitrary. Amyotrophic lateral sclerosis is described as distinct from the progressive spinal muscular atrophy.

The primary lesion of tabes dorsalis, according to Starr, lies in the posterior spinal ganglia and in the ganglia of the cranial nerves, and possibly in the ganglia of the sympathetic nervous system. This is a very positive statement, and expresses an opinion that is by no means universal. According to the figures afforded by Starr's cases, about seventy per cent of tabetic patients have had syphilis. He is unwilling to admit that there is "no tabes without syphilis." In regard to the much disputed question of antisyphilitic treatment in tabes, he advises that this treatment should be employed whenever a history of previous infection is obtained, or whenever its existence is strongly suspected.

No one doubts that chronic myelitis may develop as the result of an acute myelitis, when a certain amount of damage in the spinal cord is produced, but we have the very positive expression of opinion from Starr that myelitis may be from its onset a slow process, and thus chronic from the start.

The views concerning operation in injury of the spinal vertebræ are conservative and wise, if the reviewer may be permitted an expression of his own opinion. There should be some reason to hope for removal of compression before operation is attempted. "If there is evidence, by X-ray examination," says Starr, "of an existing compression of the cord by displaced bone, this should always be removed. If, however, there is no such evidence an operation should be refused." The reviewer has seen a person with fracture of the vertebræ, who had recovered partially in course of time, placed in a much more serious condition by exposure of the spinal cord. Nothing is said regarding operation upon the spinal column in spinal caries. Laminectomy may cause distinct increase in the severity of the symptoms in this disease, as in a case recently seen by the reviewer.

Much might be written concerning the excellent chapters on the diagnosis and localization of cerebral disease, on the cerebral atrophies of childhood, on encephalitis, paresis, abscess and tumor of the brain, etc., but this review can not be more than a brief notice of a thoroughly praiseworthy book, and a feeble attempt to show that the author speaks in no uncertain terms on many disputed subjects, and reveals a wealth of clinical and pathological observation that makes his text-book one of the best on organic nervous diseases in existence.

The temptation to mention the results of Starr's cases of brain tumors is, however, too great to resist, because it is well known that for a long period this has been one of the cerebral disorders to which the author has devoted especial attention. One must read with a feeling akin to sadness and disappointment that but nine per cent of tumors of the brain are open to operation, and that, therefore, in the vast majority of cases we cannot give the patient any hope.

Let us wish a hearty reception to this recent and most excellent text-book on organic nervous diseases. It is elaborately and judiciously illustrated. SPILLER.

A TEXT-BOOK OF LEGAL MEDICINE AND TOXICOLOGY. Edited by **FREDERICK PETERSON**, M.D., Chief of Clinic, Nervous Department of the College of Physicians and Surgeons, New York; and **WALTER S. HAINES**, M.D., Professor of Chemistry, Pharmacy and Toxicology, Rush Medical College, in affiliation with the University of Chicago. Two imperial octavo volumes, illustrated. Philadelphia, New York, London: W. B. Saunders & Company, 1903. Vol. I.

The scope of this work is outlined by the editors in their preface in the following words: "The object of the present work is to give to the medical and legal professions a fairly comprehensive survey of forensic medicine and toxicology in moderate compass. We believe this has not been done in any very recent work in English. A number of manuals of limited size and scope have been presented on the one hand, and on the other certain systems of legal medicine of almost encyclopedic dimensions. Both find fields of great usefulness; but there is still left a broad ground intermediate between the two which we trust the present work will fill, and it was in this hope that the book has been planned and executed."

The work is made up of a series of comprehensive articles on the different departments of forensic medicine by men of acknowledged ability and wide reputation, and while a detailed review of the work as a whole is hardly possible in a short space, certain of the articles merit more than passing mention.

The Introduction by the editors contains in a short space much sound advice for both expert and attorney which if more generally followed would do much to relieve expert testimony of the discredit with which it is now too often received. "The Technic of Medicolegal Postmortem Examination," by Ludvig Hektoen might well be read by any practitioner of medicine, especially the country practitioner, who may often be placed in a position where he is forced to do a postmortem for the coroner, for upon his knowledge of methods, especially in poison cases, may rest the conviction of the guilty or the acquittal of the innocent.

Articles worthy of special mention are those on "Identity," and "Sudden Death," by James Ewing; "Death and Injuries by Lightning and Electricity," by Smith Ely Jelliffe; "Wounds," by Lewis Balch; "Speech Disorders," by Frank Warren Langdon; while the views expressed in the articles on "The Stigmata of Degeneration," by Frederick Peterson, and "Idiocy, Imbecility, and Feeble-Mindedness," by Frederick Peterson and

Smith Ely Jelliffe, have already become standard through the former author's text-book of "Mental Diseases."

The article on "Insanity," by J. T. Eskridge, is especially valuable because of the short, clean-cut definitions and clear phraseology. A somewhat unfortunate typographical error, however, occurs on page 630 in the use of the word "effects" for "affects" in the following quotation: "'Wahusinn' is a mental disorder in which the delusions and hallucinations are rapidly combined into a complete whole, intimately associated with strong effects. In Verrücktheit the effects are only accidental elements of the clinical history and disappear rapidly."

An important chapter dealing with a subject quite new to works of this character is that on "The Destruction and Attempted Destruction of the Human Body by Fire and Chemicals," by Walter S. Haines. It is an excellent study founded both on experimentation and several notable cases among which the Luetgert case tried in Chicago in 1897 figures largely.

The work of the publishers has been well done, the paper and print are good, the illustrations numerous and some of them of unusual excellence. On the whole we can warmly recommend the work to those desiring light on the subject matter of which it treats, and only trust that the second volume will maintain the high standard of excellence attained by the first. WM. A. WHITE (Binghamton).

# News and Notes.

DR. F. W. LANGDON has accepted the position of visiting consultant to the Cincinnati Sanitarium since the death of Dr. Everts.

PROF. E. KRAEPELIN, of Heidelberg, has been appointed Professor of Psychiatry at Münich.

DR. ORPHEUS EVERTS, long and favorably known to American alienists, died at his home in Cincinnati on June 19, 1903, in his seventy-seventh year, after an illness of some weeks marked by general failure of nutrition.

Dr. Everts served throughout the Civil War as surgeon in the Army of the Potomac; later he was Superintendent of the Indiana Hospital for the Insane, and was one of the experts called by the U. S. Government in the trial of the assassin of Garfield. He was a welcome contributor to the medical press and to general literature. For the past twenty-four years he has been Medical Superintendent of the Cincinnati Sanitarium.

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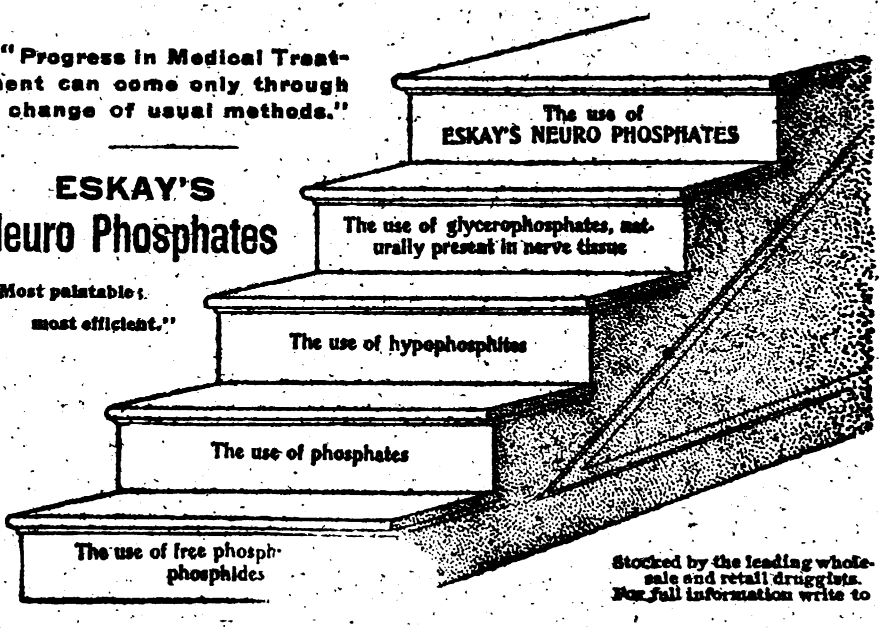
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The Official Organ of

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The New York Neurological Society

The Philadelphia Neurological Society and

The Chicago Neurological Society

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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

Printed by the Alliance Press Company, 28 W. 18th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, [illegible]

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# THE Journal OF Nervous and Mental Disease

## Original Articles.

### GIGANTISM AND LEONTIASIS OSSEA, WITH REPORT OF THE CASE OF THE GIANT WILKINS.

By Peter Bassoe, M.D.,

RESIDENT ASSISTANT PATHOLOGIST, PRESBYTERIAN HOSPITAL; ASSOCIATE IN PATHOLOGY, BUSH MEDICAL COLLEGE, CHICAGO.

The clinical features of this case have been studied by a number of prominent men both in this country and Europe in the course of the past ten years. So far as I know, the first description of the case was given by Dana, in 1893, in an article entitled: "On Acromegaly and Giantism with Unilateral Facial Hypertrophy." (13.) He first reports a case of acromegaly in an Indian 6 ft. 7 in. tall, then in introduction to this case remarks:

> "The following case seems to be very apposite in this connection, because together with a gigantic general growth, there is a special hypertrophy of part of one extremity. It might be called a case of somatomegaly, a name suggested to me by Dr. Frank P. Foster, with a hemiacromegaly of the head. In plainer language, the individual is a professional giant with an enormous special development of one-half of the face."

Dana's description is as follows: "Lewis Wilkins, aged nineteen, single; occupation, freak; was born in Minnesota. His father was a native of New York; his mother, of Canada of English stock. His parents were healthy people of average size. He has six brothers and sisters, all of ordinary height. He was the second child. He was always large for his age, though not remarkably so. He grew steadily, however, until by the age of seventeen he was over seven feet. He is now nearly twenty years old, and has grown one and a half inches in the last year. His present height is seven feet four inches, his weight three hundred and twenty-five pounds. His general proportions are for the most part good, but his feet and hands are particularly enormous, and the left side of the face shows a remarkable osseous hypertrophy, involving the frontal bone, upper and lower jaws. The hypertrophy corresponds closely with the distribution of the left trigeminal nerve. It gives his face a curious twisted and asymmetrical look, which is shown imperfectly in the photograph. The first impression is that he has a right hemiatrophy of the face. Closer inspection reveals, however, an enormous thickening of the left upper alveolar processes. The bone bulges out above the teeth as though he had a gumboil. The palatal arch is also greatly enlarged on the left side. The lower jaw is less affected, but is larger and longer on the left side. The teeth are white and even, and are not enlarged. The orbits are alike, but the left brow, and, indeed, the whole frontal bone bulges out so as to give a curious deformity to the skull. The thickening reaches back as far as the coronal suture and stops there. The head in brief is large, measuring 65.5 cm. in its greatest circumference. The osseous hypertrophy makes the naso-bregmatic arc very large, vis., 18.5, while the binauricular arc, measured through the bregma, is relatively smaller, as it does not go over the hypertrophied area. The measurements are: Greatest circumference of head, 65.5 cm. (25¾ in.); naso-occipital arc, 43 cm. (17 in.); naso-bregmatic arc, 18 cm. (7⅛ in.); binauricular arc, 37 cm. (14½ in.); from angle of jaw to symphysis of chin, right side, 13 cm. (5⅛ in.), left side 18 cm. (7⅛ in.), a difference of 2 inches. The circumference of the chest at the mammary line was 47½ in. and the expansion 3 inches. This shows that he has a thorax of not excessive size proportionately. It is a good deal smaller than that of the Indian giant, whose height was 6 ft. 7 in. and whose chest measured 50 in. The hands were enormous, measuring 26 cm. (10¼ in.) from the tip of the middle finger to the process of the ulna; the circumference of the open hand around the middle of the palm, 27 cm. The feet are relatively still larger. He wears a shoe just a foot and a half long, while the actual total length of each foot is 35.5 cm. (14 in.), and the circumference around the instep is 29.2 cm. (11½ in.). There is no especial asymmetry of physique except in respect to the face, as described. The left shoulder, however, is a little higher; he is decidedly round-shouldered, and there is a slight dorsolumbar lateral curvature of the spine. He has no cutaneous eruptions, no pigmentation or discoloration. He has thick, coarse hair, but no beard. His muscular system is but moderately developed: the grasp of his hand is weak; he does not like to climb stairs; he has not much strength. He has good co-ordination; is a good shot. His knee-jerks are slow and feeble. Vision is good in both eyes, and he has no contraction of the visual field. The pupils react normally. The eyes are small, the palpebral fissure measuring 3 cm. His intelligence is good. He sleeps well and eats well. He has a prodigious appetite, and on one occasion ate 27 plates of ice cream at one sitting, thereby winning a wager that he could eat more than two men. No unpleasant after-effects were reported. He has slight headaches at times. His pulse beat and respiration and his heart action were normal. I could not say whether the thyroid was changed in size. It is certainly present." Dr. Dana, in conclusion, remarks: "The interest of this case lies first in the giant growth, and next in the progressive facial hemihypertrophy. That giantism is sometimes associated with acromegaly has been shown by my own case and that of others cited. This patient has some symptoms belonging to acromegaly, viz., the enlargement of the bones of the left side of the face, beginning at about puberty, the kyphosis and scoliosis, the enormous feet, the coarse hair, feeble muscular development, and prodigious appetite. The progressive facial hypertrophy is very interesting on account of its rarity and its association with giantism."

On April 24, 1896, Mr. Wilkins was presented to the Vienna Medical Society by Lamberg. The proceedings as given by the *Wiener klinische Wochenschrift* (29) were as follows:

"Lamberg presented Wilkins. The man, age twenty-two years, has healthy parents. Growth was normal until the fourth year. Then he grew rapidly until the sixteenth year. At eighteen he reached the present height, 245 cm. He is 20 cm. taller than the giant skeleton in the Vienna Anatomical Museum. He presents some skeletal abnormalities, viz., scoliosis, asymmetry of the pelvis, and a tumor of bony hardness, originating from the left upper jaw, which perhaps also causes a narrowing of the left nasal cavity. There is also considerable swelling of the left half of the forehead. The intelligence is proportionate to his age. The genital functions are normal. Lamberg considered the case as one of acromegaly.

"M. Sternberg: Only three skeletons in museums are larger, the Irsh giant at Dublin (259 cm.), one at St. Petersburg (254 cm.), Patrick O'Byrne, London (249 cm.). Giants frequently present morbid features, general dystrophies or vegetative disturbances. About 40 per cent have acromegaly. Doubtless there are also quite normal giants. He does not consider Wilkins a case of acromegaly. The prominence of the lower jaw is lacking as well as deformities of the soft parts of the mouth and nose; the tongue is normal, the hands are proportionate, etc. Abnormal is the great development of the left half of the face. The lips are also somewhat thickened on the left side, so we may speak of a hemihypertrophia facialis, principally ossea. The deviation of the axis of the left eyeball and the narrowing of the left nasal cavity point to bony changes in the orbit, and the slowness of movements and phlegmatic temperament to intracranial hyperostoses and exostoses. Buhl's case, Hassler, is probably the most analogous of the known cases.

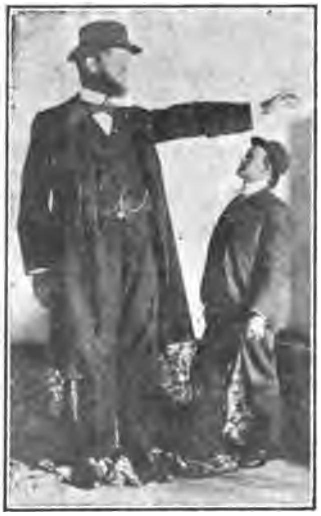

Fig. 1 Mr. Wilkins and his brother.

"Schlesinger accentuated that acromegaly was clinically sufficiently sharply limited to exclude this case. Above all, the enlargement of the soft parts, almost always present in acromegaly, was lacking. The lips and tongue are approximately normal. So is the position of the teeth, which is almost always altered in acromegaly on account of the enlargement of the lower jaw. Schlesinger asked about the eye findings, as ocular disturbances are frequent in acromegaly, such as bitemporal hemianopsia, optic atrophy and paralysis, due to the frequency of hypophysis tumors.

"Lamberg: 'The eye findings are normal.' "

In his exhaustive treatise in Nothnagel's "Spec. Pathologie," Sternberg (43) again discusses this case and groups it together with Buhl's and one of Sirena's as "pathological," non-acromegalic gigantism, the "pathological" features in this group being tumor-like exostoses (leontiasis ossea and hyperostoses). In connection with leontiasis he says (p. 86): "If the disease is extensive and associated with gigantism, a confusion with acromegaly is likely, as actually happened in the case of the giant Wilkins (Lamberg). Here was to be considered that the enormous hands and feet were proportionate to the excessive size of the body, and that a close examination showed the presence of bone tumor, with absence of acromegalic changes of bones and soft parts."

Schmidt, in his treatise on gigantism (41) takes the same view of the case, being positive that this case is not one of acromegaly, but analogous to that of Buhl. The latter case will be considered in detail later.

Mr. Wilkins was admitted to the service of Professor Bevan at the Presbyterian Hospital, June 28, 1902. The writer wishes to thank Professor Bevan for his kindness in allowing him to use the following history, which was obtained by the interne, Dr. Pearsall:

Lewis Wilkins, age 28. Family history: Father, age 55; mother, age 60; both well. Four brothers and one sister alive and well. All relatives and ancestors, so far as known, have been of normal stature. No history of tuberculosis or tumors in the family.

Personal—Patient weighed 9 pounds at birth, and was normal in size until four years of age, when he began to grow rapidly, and at ten years was over 6 ft. in height, and continued to grow until about 18, when he attained his present height (8 ft. 2 in.). He has been on exhibition the past twelve years, and has traveled extensively in the United States and Europe.*

Usual diseases of childhood. Malaria one year ago. Lateral curvature of spine for about twelve years. When eight years old he noticed a growth, size of a small marble, above the left eye. This has continued to grow slowly until four or five years ago, since which time he thinks it remained stationary until three months ago, from which time it has increased in size rapidly. About two years ago, he states, he saw Professor Virchow in Berlin, who, so he says, pronounced the growth a benign one, said it was on the external surface of the skull and would probably never cause him trouble. Gonorrhea four years ago, discharge lasting about three weeks; denies other venereal infection. Three months ago he would have severe pain in left frontal and temporal region each morning on rising, pain disappearing gradually by 10-11 o'clock. For the past month he has had continued pain in this region when awake and has required the constant use of opiates. About one month ago he lost the vision in the left eye. Thinks it has been weak for several years. One week ago he also lost the sight in the right eye. Says vision disappeared suddenly; saw dimly on retiring one evening and the next morning could not appreciate light. He has had several spells of vomiting during this time. These spells have not been closely connected with eating. He states that he has had no sensation over the left side of the face for about one month, and during this time has had difficulty in speech, his voice being thick and heavy. Two months ago he experienced ringing sensations in the left ear and has since complained of earache. Says he is deaf in the left ear. Since his arrival at the hospital he has been dull; almost semi-stuporous. Has had several involuntary bowel movements. He was taken with diarrhea while on the way to Chicago from his home in Oklahoma.

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*In a pamphlet distributed at Mr. Wilkins' exhibits in Germany it is stated that he did not grow in height from the age of 18 to 21, but gained 3 inches from 21 to 22, when he reached 245 cm. It is also stated that his father measured 161 cm., his mother 170 cm. in height.

Physical Examination. Height 8 ft 2 in., according to statement of patient. Measures 56 in. around the chest. He is well proportioned. There is a growth on the left side of the head which extends from the median line outward and backward as far as the external auditory meatus and downward over the face to the alveolar process of the superior maxilla. This mass is firm except in an area a little larger than a silver dollar in front of and above the external auditory meatus which is soft and semi-fluctuating. The left eye is closed. The left upper lip is thicker than the right and droops. The pupils react neither to light nor distance, and the left eyeball is immobile. The tongue does not deviate on protrusion, and the muscles on both sides act in frowning or laughing. There is an area of anesthesia extending from the left ramus of the lower jaw to the external auditory meatus, and upward to the median line of the skull. The left half of the tongue is also devoid of sensation except for a narrow strip near the median line. 6-28, urine 1027. Acid. No albumin or sugar. No casts. Red cells and leucocytes present. 6-28, blood examination, hemoglobin 85 per cent; reds, 4,876,000; leucocytes, 9,300. Temp., 98.4-101.4; pulse, 80-92 on day of admission. 7-1, urine, 2,400; cc., 1,010, ac. No albumin or sugar. Urea, 1.75 per cent; temp., 98-100. Ophthalmoscopic examination showed a bilateral choked disc.

The diarrhea became more severe while in the hospital, the stools were slightly bloody, pain and tenesmus were present. He gradually failed, and died July 10, 1902.

The autopsy was held immediately after death by Professor Hektoen, assisted by Dr. L. Loeb and myself. A relative who was present did not permit any measurements to be taken. Fortunately those taken by Dana nine years ago give us the relative proportions. A growth in height has undoubtedly taken place since that time, as Lamberg's measurements and the patient's statements show; also an increase in the circumference of the chest. The time allowed for the autopsy did not permit a more detailed description of the external appearance of the body than that given in the clinical notes.

After the removal of the thoracic and abdominal viscera both carotids were injected with 4 per cent formaldehyde solution in order to harden the brain in situ.

Anatomic Diagnosis.—Necrotic and ulcerative colitis and ileitis; cirrhosis of liver; chronic catarrhal gastritis; epitheliosis of esophagus; hemorrhagic broncho-pneumonia (right lower lobe); enlargement of thyroid; sarcoma in region of hypophysis; extension of tumor to subcutaneous tissues; diffuse hyperostosis of frontal, left parietal, left temporal, and left superior maxillary bones; calcification of left pleura and spinal arachnoid; compression of brain; general gigantism.

The abdominal cavity is empty and the peritoneum smooth and free from adhesions. The diaphragm reaches to the fourth interspace on both sides.

Both pleural cavities are empty and there are no pleural adhesions, except between the right upper and middle lobes.

The pericardium is smooth and the cavity contains no fluid.

The thyroid weighs 112 grams; it is uniformly enlarged, and glistening on the cut surface as if the follicles were dilated with an unusual amount of colloid material. In the left lobe are two small, circumscribed, somewhat reddish nodules.

There is no trace of the thymus in the corresponding fatty masses.

The larynx and trachea show no changes.

The lungs are crepitant throughout, much lighter and spongier than usual, especially in the case of the left lung. In the lower right lobe are two hemorrhagic areas with elevated bases, the longest diameter being 6 cm. In the pleura over the lower left lobe is a flattened calcareous district, 1 cm. square, and at the right of the left lung are calcareous glands.

The heart is normal in shape and weighs 465 grams. There are no changes in the endocardium. The myocardium is firm and reddish brown.

The aorta is quite smooth throughout its entire extent.

The spleen is large and its capsule smooth. Cut surface is reddish and of normal appearance. The spleen weighs 620 grams.

The lymph nodes in various parts of the body are not enlarged.

The pharynx is normal.

The esophagus shows a number of flattened, whitish, firm, raised, nodular areas in the upper part of its course, generally arranged somewhat linearly. Otherwise the membrance is smooth.

The stomach is very large. It weighs, including the duodenum, 655 grams. The mucous membrane is thrown into huge wrinkles, not unlike cerebral convolutions, which cannot be straightened out on stretching the muscular coats. The mucous surface is covered with a turbid, tenacious mucus.

The small intestine from the duodeno-jejunal junction to the ileo-cecal valve, measures 20 meters in length. The mucosa is normal in appearance until within about 50 cm. of the valve, where it appears granular, rough, and of a dirty greenish yellow color in areas, with rather superficial small ulcers. Just above the ileocecal valve the entire circumference of the ileum, for about 5 cm., is intensely necrotic.

The large intestine is very voluminous; it measures in length, approximately, 4 meters. Its entire mucous membrane from the ileo-cecal valve to the anus is the seat of necrosis in the form of grayish, dirty, granular points and areas, and more especially of thickly disseminated, rather small ulcers with smooth floors as a general rule, and overhanging margins. In many cases the undermining of the margins is so extensive that large submucous tunnels are formed and neighboring ulcers may communicate with one another by means of these passages. In the flexures, and especially in the rectum, congestion of the mucous membrane is noticed.

The liver is large, weighing approximately 4,000 grams; it has a granular and nodular surface, especially near the lower margin of the right lobe. There is some increase in consistency and more or less distinct breaking up of the surface into large islands of liver tissue by fibrous bands. The under surface of the liver shows anomalous formation of the lobes, the right being taken up largely by a large projecting mass with some constriction at the base of its junction with the main part of the liver.

The gall bladder and bile passages do not show any changes.

The pancreas is large, weighing 275 grams; it is normal on the cut surface.

The ardenals are large, weighing 42.5 grams; they do not show any changes on the cut surface.

The kidneys also are large, weighing together 525 grams.

Capsule is free except over a rather large area in the left kidney, where there is an irregular, radiating, depressed scar. The surface is, in general, smooth. The cortex is unusually wide; the cortical markings distinct, and the glomeruli visible as reddish points. In the apices of several pyramids are small deposits of a granular, sand-like material.

The mucous membrane of the ureters and of the bladder is smooth. The bladder is distended with urine and is very large.

The prostate is not as large, in proportion, as other organs. The same seems to be true of the seminal vesicles and testicles, the latter weighing 85 grams.

There is a soft subcutaneous nodule almost as large as a fist behind the left ear; and another of greater firmness involving about two thirds of the left side of the forehead. The tumor tissue on section is pink and homogeneous. The frontal region of the skull is very thick, especially on the left side. The skull cap weighs 1,450 grams; it is 68 cm. in circumference, 25 cm. in length; from the obliterated interfrontal suture, lower end, to the right coronal suture, internal horizontal measurement, is 5.7 cm.; from the same point to the left coronal suture is 8.5 cm. In the median line the frontal bone is 8.5 cm. thick, and is softer than normal, especially on the left side. There is marked asymmetry, the interparietal suture being displaced to the right. Fissures for vessels are relatively shallow. In front of the coronal suture the

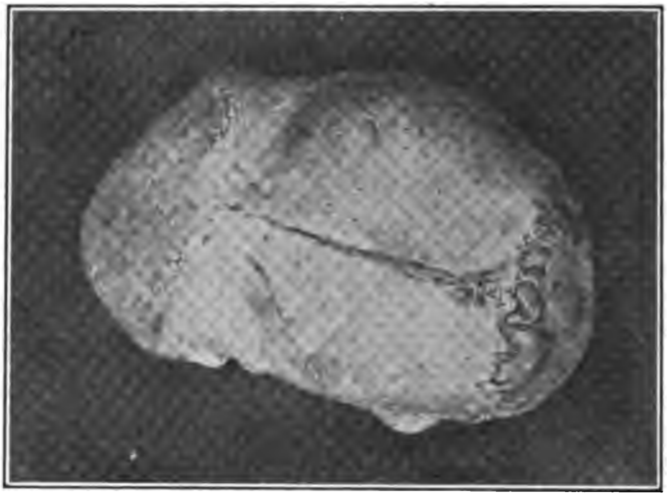

Fig. 2 Outer surface of skull-cap.

bone is rough from the presence of thickly-set depressions, varying from 0.5 to 3.0 mm. in diameter. The remainder of the skull cap is smooth. The thickness varies from 2 to 8 mm. On the outside the interparietal suture presents a prominent ridge in its posterior half (see photograph). The parieto-occipital suture is also prominent. The base of the skull is greatly deformed. On the left side the anterior fossa is obliterated by the thickened frontal bone, and the middle fossa also is almost filled in with bone.

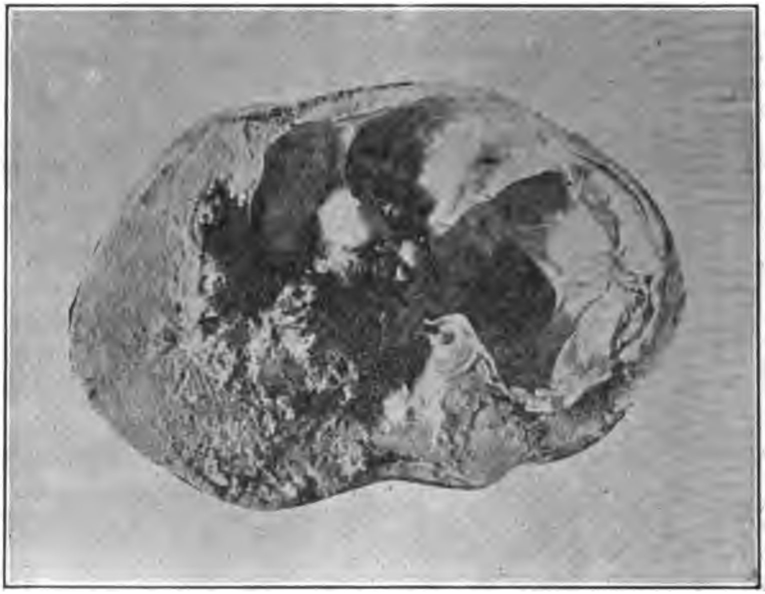

Fig. 3 Base of skull.

The sella turcica is wide, its floor partly eroded. In this region is a large tumor mass which has grown into the pharynx, orbits, and ethmoidal sinuses, and has destroyed the roof of the nose; the roof of the left orbit has been much more extensively invaded than the right. In the median line of the surface of the tumor exposed at the base of the skull after removing the brain is a projecting pedicle, darker than the tumor mass. This is found to be the infundibulum. It measures 3 mm. in diameter and is inserted into a round elevation 13x6 mm. of the same color as the stalk. The tumor itself is white on the surface, lobulated, the lobules 2 to 4 mm. wide, separated by narrow, but deep, fissures. On section the superficial part of the tumor is white and distinctly lobular. A gelatinous fluid exudes from the cut surface. Deeper down it is more homogeneous and there is more slimy fluid. Still deeper dark red bands permeate it. The portion of the tumor removed with the hypophysis weighs 150 grams, but at least as much more was scooped out in pieces or left behind.

The brain weighs 1,540 grams. The distance from the frontal to the occipital pole is 18 cm.; transverse diameter, 14.5 cm. There is marked deformity of the left hemisphere to correspond with the obliteration of the anterior fossa of the skull. The left frontal lobe has been pressed upward and to the right. It terminates in a narrow strip along the median fissure. The lower end of the strip is 8 mm. above the pole of the right frontal lobe, its width 15 mm. at the base, 8 mm. at the lower end (see photograph). Farther to the left the brain substance has been compressed so that the inferior surface of the left frontal lobe is 3.5 cm. higher than that of the right side. The left temporosphenoidal lobe also is smaller and compressed; its inferior surface measures 1.5 cm. antero-posteriorly, while the right measures 3.7 cm. The convolutions of the brain are generally broad and flattened; the sulci shallow (see photograph). The left optic nerve is narrower than the right. The other cranial nerves appear symmetrical.

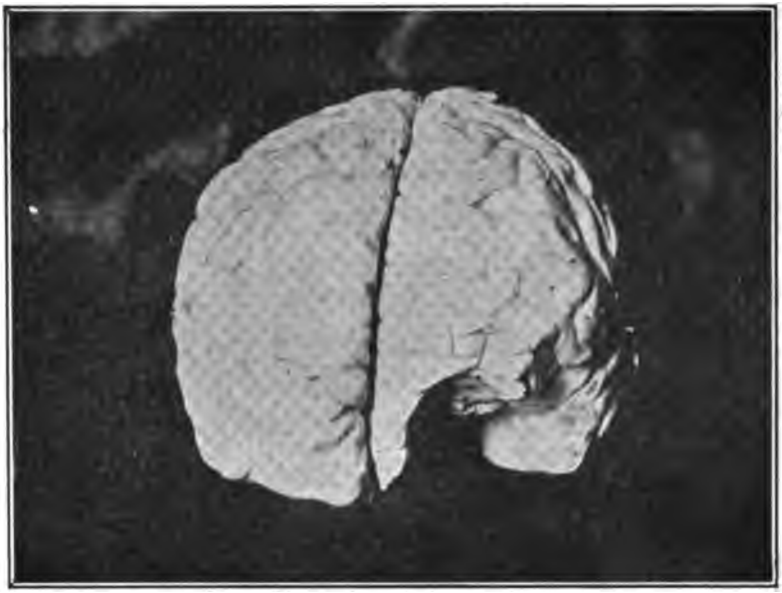

Fig. 4 Anterior view of brain.

The length of the medulla and cord, from the pons to the filum terminale, is 60 cm. Below the mid-thoracic region are many thickened, partly calcified patches on the inside of the dura. These are rough and granular on the inner surface; the attached surface, on separation from the dura, is smooth. The largest of them measures 3x1.2 cm. To one of them a nerve-root is adherent.

The sternum is 26.5 cm. long, the greatest width of the manubrium 10.5 cm., of the body, 5.5 cm.

Histological Examination. The soft tumor. All parts of it were subjected to examination, and its structure was found to be essentially the same everywhere. It is made up of cells, mostly spindle-shaped, with large oval or round nuclei. Karyokinetic figures are numerous. The protoplasm of the cells stains faintly. There is no suggestion of glandular or other epithelial structures, the cells having no definite arrangement. The texture is loose, there being a large amount of finely granular intercellular substance, stained lightly by hematoxylin. This is evidently in part the coagulated product of the abundant viscid material noted on gross examination. In the deeper part of the tumor the degenerative changes are more marked. There are large necrotic areas,

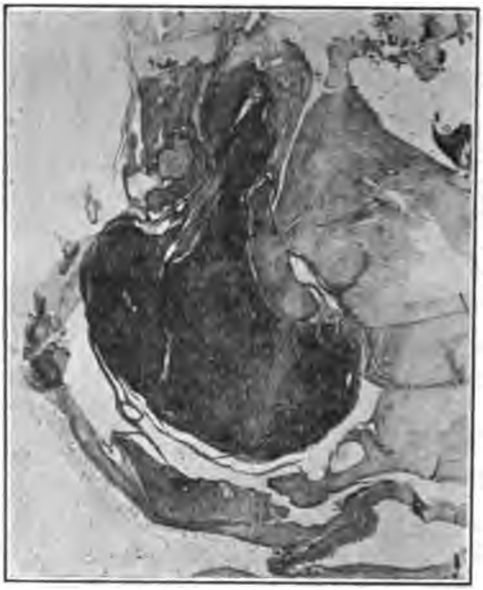

Fig. 5. Magnified 8 diameters. Showing relation of hypophysis to surrounding tissues, mostly tumor.

and also hemorrhages. The reason for the lobulated appearance of the surface is found in the presence of fibrous bands running perpendicularly to the surface. Such trabeculæ are also seen in the deep portions. In places tumor cells, sometimes with karyokinetic figures, lie in them. In various parts, more particularly near the hypophysis, are bony spicules, some of which are rich in bone corpuscles, and probably newly formed. Here and there giant cells are seen. The tumor may be designated an osteoplastic sarcoma with edema and mucoid degeneration.

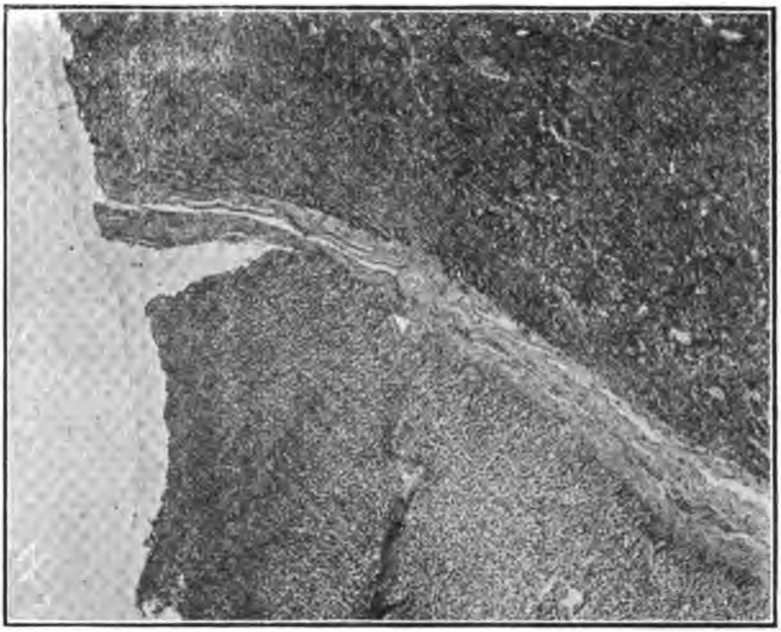

Fig. 6. Magnified 45 diameters. On one side of dura hypophysis tissue. On other side tumor with bony spicule.

The Hypophysis and its Relation to the Tumor. Serial sagittal sections* were made of the lateral halves after a preliminary bisection through the infundibulum. A fibrous membrane (dura mater) was found separating the hypophysis from the tumor, the sharp border being plainly visible to the naked eye on account of the difference in staining. At one point on the left side the tumor and the hypophysis are in direct contact, but the border is sharp, and there are no transitional cell forms. Some enlargement of the organ is present, the greatest length as seen in the sections being 20 mm. At this point it measures 8 mm. from above downwards. It appears flattened in all the sections. The histologic structure is essentially normal. There are the usual large, highly eosinophilic cells, arranged in elongated columns, or in clusters of about the size of renal glomeruli, and smaller cells with cytoplasm stained a faint blue with hematoxylin, and rounded nuclei, also with more or less glandular arrangement. In the extreme right portion of the anterior lobe this is the only cell type seen, and glandular arrangement less apparent. There is a moderate number of spaces containing a colloid material and lined by epithelium. Engorged vessels are abundant. There are also numerous hemorrhages, a few foci consisting of densely-packed, small, round, deeply-stained mononuclear cells, an occasional amyloid body, and in places the large eosinophilic, epithelial cells are vacuolated. In places the trabeculæ and adventitia of the small vessels have a hyaline appearance. There are generally deposits of pigment at the margin of such areas, indicating that they probably mark the site of old

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*The writer wishes to thank Messrs. Foreman, Jackol, and Darmer for valuable aid in this tedious work.

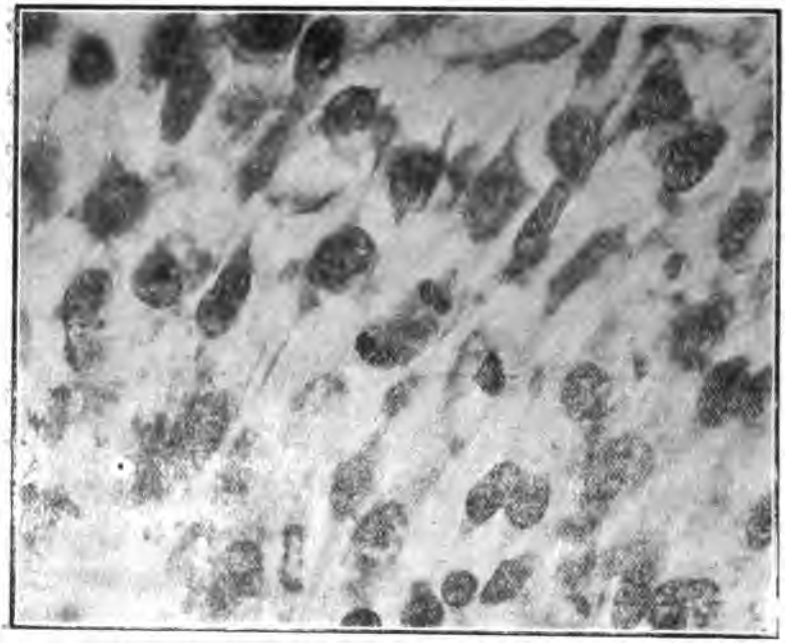

Fig. 7. Magnified 1,200 diameters. Showing finer structure of tumors. One mitotic figure in the center.

hemorrhages. The posterior lobe is small and made up of a faintly fibrillar ground substance, stained lightly by hematoxylin, and containing a moderate number of long and narrow, deeply-stained nuclei; small vessels are numerous and engorged.

The Skull. Section from frontal bone in median line: Less than half the section is compact bone in irregular islands, the remainder a loose reticular tissue, consisting of a fibrillar unstained matrix with a small number of long, narrow, nucleated cells. At one edge of the specimen the interstices between the islands of bone are densely packed with spindle-cells like those in the soft tumor.

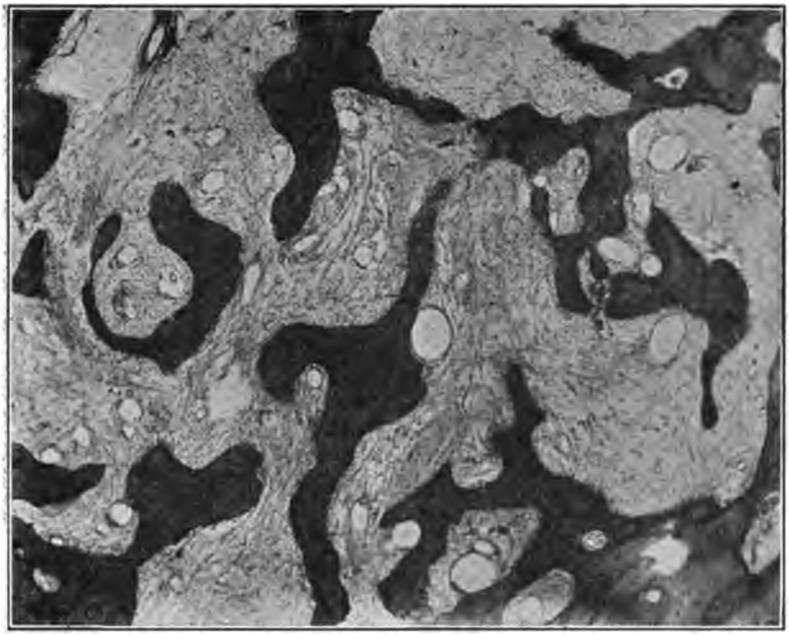

Fig. 8. Magnified 38 diameters. Frontal bone in median line.

Left side of frontal bone near external surface: Here the compact bone is even smaller in amount, lamellæ imperfect. Haversian canals large and numerous. Most of the bone appears to be of recent formation judging from the number and size of the cells. Abundant osteoblasts are seen, mainly consisting of spindle-cells, with frequent mitotic figures. The texture of the tumor tissue in places is very loose, suggesting mucoid degeneration or edema.

Section from inner surface of frontal bone differs from the preceding by the larger amount of deposits of new-formed bone, rich in cells, and taking a deeper hematoxylin stain than the old bone. They form small round or irregularly shaped bodies, lying together with spindle-cells, osteoblasts, and certain large, faintly-stained oval cells in the spaces bounded by trabeculæ of old bone. In notches in the surface of the latter osteoclasts are occasionally seen. Right sphenoid bone. Here the bony trabeculæ are also scarce, and enclose tumor tissue everywhere. In places are islands of new-formed bone surrounded by osteoblasts and a few osteoclasts. Sections from a rib show nothing abnormal.

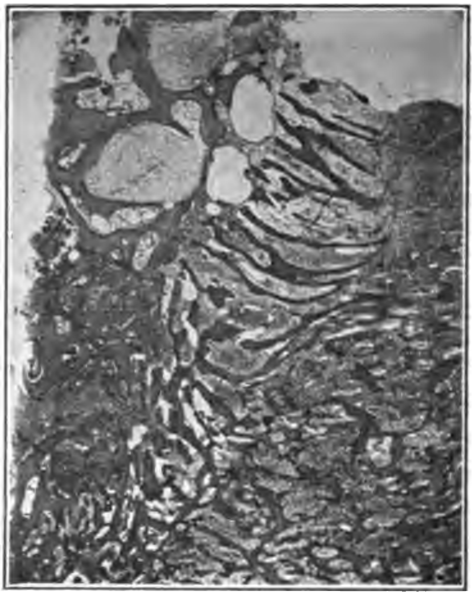

Fig. 9. Left side of frontal bone, showing sarcomatous invasion.

The ganglion cells of the cerebral cortex are practically normal. A few of them contain yellow pigment with lateral displacement of the nucleus. Marchi preparations of the cortex and subcortical white matter show no degeneration. There is a moderate amount of diffuse degeneration in the lower medulla and cervical cord. The roots are also slightly affected. In the cervical spinal ganglia a number of amyloid bodies are seen, the vessels of the capsule have thickened hyaline walls, a few of the small vessels contain thrombi. Many of the ganglion cells contain large masses of yellow pigment. The nuclei are distinct, generally centrally located; their staining is very variable in intensity. There is some thickening of the smaller arteries in the lumbar region of the cord. The calcified patches in the spinal meninges are found to contain true bone. Both optic nerves are extensively degenerated, more so the left, as seen in Marchi preparations.

Thyroid. The acini are generally distended with homogeneous colloid material. In places several acini appear to have coalesced. The blood vessels are few and small.

Lung. In a section from the lower lobe of one lung the alveoli are completely obliterated, so that it is difficult to recognize lung structure. In most places the lung tissue is replaced by round and elongated connective tissue cells. Here and there are considerable areas of blood which is partly in blood vessels and some apparently free in the tissues. The connective tissue is well organized into strands in places; in other places the organization has not gone so far. In one small area the alveolar walls may be made out, and the centers of the alveoli are filled with red blood corpuscles and leucocytes. Developing capillaries are seen in the tissue. These are filled with corpuscles. There are some pigment patches near the blood vessels. The pleura is thickened and greatly congested.

Heart. In sections of the heart muscle there is seen a very slight increase in interstitial tissue; otherwise there are no changes.

Spleen. The spleen is much congested, red corpuscles being diffused quite uniformly throughout the tissue. Malpighian corpuscles are clearly marked.

Lower Ileum. In sections examined, the mucosa is absent. The submucosa is greatly infiltrated with round cells. Blood vessels are congested, and in some places there is hemorrhage into the tissues. In places the submucosa has undergone necrotic change and sloughed away down nearly to the muscular layer. The circular layer of muscle fibers also is infiltrated with round cells and in many places there are blood vessels distended with blood. In the submucosa can be seen thrombi, consisting of layers of fibrin, between which are red and white corpuscles. Fibrin also is deposited in shreds throughout the submucosa.

Colon. The mucosa is much eroded in most places, and in some entirely absent. The surface is necrotic, so that only the lower parts of the glands are seen. The lumina of many of the glands contain mucous material. There is considerable round-cell infiltration. There is also hemorrhage into the tissues, and the vessels are greatly engorged. This condition extends also in the submucosa as far down as the muscular layer. Throughout the submucosa there are shreds of fibrin arranged usually in an irregular network. Near the mucosa an amœba may be seen occasionally.

Rectum. The rectum presents much the same appearance as the colon, except that the condition is much more marked. The mucosa has undergone necrosis, and sloughing has occurred. There are large spaces near the surface, some of them filled with blood and fibrin; others are empty. In some places the blood is confined to blood vessels, but frequently it is diffuse. There are numerous round cells, some polymorphonuclear leucocytes, and plasma cells. Amœbæ are present in large numbers, most abundant in the upper part of the submucosa, but are also found in the other coats. They are usually spherical, but occasionally oval or oblong. They stain with eosin faintly. Usually the nucleus can not be seen, but frequently it is quite distinct, though not stained with hematoxylin. The amœbæ are invariably vacuolated. They are found mostly in the solid tissues, but occasionally may be seen in the blood vessels or large lymph spaces.

Liver. Interlobular connective tissue is increased in amount and distinctively isolates the liver lobules; this connective tissue does not extend into the lobules. The bile capillaries and blood vessels in this tissue show no changes. At the margins of the lobules the liver cells are not as distinct as elsewhere, and seem somewhat compressed. Elsewhere the liver cells are normal and the nuclei distinct. Occasionally fat vacuoles may be seen. The capsule of the liver is considerably thickened.

Pancreas. The pancreas shows no changes. Numerous areas of Langerhans are present.

Adrenals. There are a few areas of round-cell infiltration in the medulla and in the zona reticularis. Under the capsule in the cortex, there is seen congestion of the blood vessels.

Kidney. The glomeruli completely fill their capsules in most cases. Many of them are congested and show round-cell infiltration. The capsule is somewhat thickened. In places the capillaries between the tubules are much congested; there are also many round cells in places. The renal cells are large, cell walls distinct, and nuclei clear and distinct. In the tubules is seen a small amount of granular material. In some places the cells lining the tubules are slightly swollen and granular. Frequently clear spaces are seen around the nuclei. In other tubules the lining cells are low and flat, the nuclei stain deeply, and the lumen of the tubule is filled by a granular material.

Testicle. The seminiferous tubules show quite marked changes. Just inside the basement membrane the sustentacular and spermatogenic cells have undergone a hyaline change. In some tubules the hyaline change has advanced to the center, while in others the hyaline area separates the normal cells from the basement membrane. Some tubules are apparently normal. There is some increase of fibrous tissue, and a few areas of round-cell infiltration.

A *bacteriological examination* was made by Mr. E. H. Ruediger of Rush Medical College, who kindly reported that no growth was obtained from the heart's blood, and that two organisms were isolated from the intestinal ulcers, one a typical colon bacillus, the other an unidentified bacillus, which possessed some characteristics both of the Shiga bacillus and the ordinary colon bacillus.

A *chemical examination* of the thyroid gland was made by Dr. H. G. Wells, who kindly furnished the following report:

"One-half of the gland was received in alcohol, with the statement that the gland was symmetrical. The tissue was ground up finely in a meat chopper, and to the ground material was added the alcohol in which the tissue had been preserved; the whole was then evaporated to dryness on the water bath, and then dried to a constant weight over sulphuric acid. The total weight of the dried gland so obtained was 13.80 grams, giving a total for the entire organ of approximately 27.60 grams. The normal weight of dried thyroid glands of residents of the United States has been found to be in the vicinity of 5 grams.* Portions of the dry powder were then analyzed by the modification of Baumann's method, and the average amount of iodin of four determinations found to be 2.28 mg. per gram of dried substance. The total amount of iodin in the entire gland was, therefore, 62.928 mg. Analysis of the glands of a series obtained in Chicago showed the average amount under normal conditions to vary from 1.5 to 2.5 mgs., with a total iodin content of about 10 mgs. Similar results were found with glands removed from residents of other parts of the United States. It is seen, therefore, that the gland of the giant contained a normal amount of iodin in each gram of gland substance, but on account of its greatly increased size the total amount was six times as much as normal.

"Histological examination of the gland explains fully the analytic results. It is seen that the enlargement is of the type of simple colloid goiter, characterized by large aveoli distended by a normal colloid material. It has been shown by Wells (*loc. cit.*) and corroborated by A. Oswald,** that in goiters as a rule the amount of iodin is in direct proportion to the amount of colloid. In the goiters of the "parenchymatous" type characterized by a great hyperplasia of epithelial cells, and containing but little colloid, there is a marked decrease in the amount of iodin in each gram of gland, corresponding to the small amount of colloid, but the total amount of iodin is about normal because of the great size of the thyroid. On the contrary the glands of which this is a type show a colloid containing about normal quantities of iodin in each gram, and consequently with a greatly increased total amount. When the amount of colloid is very large Ostwald has found that part of it may not contain iodin. It seems possible that the parenchymatous goiters may be the result of a deficiency in supply of iodin in a condition to be transformed into thyroiodin, so that the gland hypertrophies in the effort, which is apparently successful, to obtain enough iodin for the requirements of the body. The colloid goiters may result from an increased requirement for thyroiodin in the economy, with normal supply of iodin, leading to the formation of much colloid with a normal proportion of iodin so long as the supply is adequate. That this giant, particularly with a diseased hypophysis, should have a large amount of iodin in his thyroid was to be expected and agrees with the theory. The result also shows that in this case of giantism the goiter was not different from simple goiter, at least so far as histology and iodin content indicate."

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*"The Physiology and Therapeutics of the Thyroid Gland and Its Congeners." H. Gideon Wells, Journ. Amer. Med. Assoc., Act. 30, Nov. 6 and 13, 1897.

*A. Ostwald, Virchow's Archiv., 1902, 169, p. 444.

(*To be continued.*)

# STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL HUMAN BRAIN AND IN DEMENTIA PARALYTICA[1]

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*Introductory.* The following contribution to the study of the cerebral cortex in the normal human brain as compared to that in dementia paralytica, does not include much that is new to the pathology of the latter, but aims to present the subject in a somewhat different manner than has heretofore been done. The writer's perusal of many anatomical and pathological articles has often been unsatisfactory to himself, owing to many generalized statements of conditions found without any adequate illustrations of what was really to be seen. It has been the endeavor in this article to present and describe photomicrographs of actual sections from a practically normal human brain histologically by way of comparison, and to follow these by photomicrographs of sections of a brain of a well marked case of dementia paralytica. These photomicrographs have been supplemented by drawings. The possibilities as well as the limitations of photomicrography in preparations stained by the Nissl method, it seems to the writer, are fairly well brought out in this article, and this was one of the objects sought in its presentation. That photomicrographs show structure and arrangement in but one plane of limited thickness is true. It is also true, however, that they show exactly what is there and the exact relation to other parts, and there is no personal equation such as is bound to occur in any drawing. A drawing, on the other hand, is always more or less composite, does not show exact relationship, and involuntarily the point to be illustrated is made to assume undue prominence. In work of this kind artefacts are constantly to be looked for and avoided, and the entire process of preparation, as well as the history of the case, must be also carefully considered before any conclusions should be drawn.

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[1] Submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy in the Faculty of Pure Science, Columbia University.

When it is considered how extremely thin the human cortex is, 1.5 to 3.5 millimeters; that its exterior surface is estimated at about 200,000 square millimeters; that it is intricately arranged in convolutions of varying size and direction; that it is exposed to injury prior to death, at the autopsy, during transportation, and also in the various manipulations of fixation, imbedding, sectioning, staining, decolorizing and mounting, it can readily be seen how artefacts may result by the time the specimen is prepared for examination. This was forcibly brought to the notice of the writer in the examination of some sections but a short time ago. Owing to a dull microtome knife the nuclei of a number of the cells were completely carried outside of the cell-body and the cell-bodies themselves broken and cracked in places. This was at first regarded as a pathological process until attention was called to the fact of the nuclei all being extruded in the same direction, and the breaks and cracks presenting a ragged and irregular appearance. Even when the greatest care is used there will be a variation in thickness in different sections—one being somewhat thinner and the next correspondingly thicker than actually registered by the microtome, although most carefully imbedded, so that variations in the thickness are also to be considered. Furthermore, in fixation, shrinkage or swelling may occur, the latter being demonstrated by Donaldson (1891), who placed fresh nerves in a solution made up of 2.5 per cent of potassium bichromate, plus one-sixth its volume of 95 per cent alcohol for three weeks. He then washed them for one day in water and placed them in 95 per cent. alcohol for three or four days, and finally in 80 per cent alcohol, using six olfactory bulbs, six olfactory tracts, and three pairs of optic nerves of the sheep. This was exactly the same treatment to which his specimens of human nerves had been subjected. The volume of specimens of olfactory bulbs subjected to this treatment and afterwards placed in 80 per cent alcohol was found to be 5.2 per cent greater than fresh specimens, those of the olfactory tracts 8.8 per cent greater, while the optic nerves showed but 2.6 per cent increase in volume. Imbedding in celloidin did not affect the volume, while some stains, as acid fuchsin, produced no change, whereas Delafield's hematoxylin caused swelling, and thus increase in volume. Hammarburg (195) found by experimenting with small blocks of cortical tissue that by accurate measurements blocks hardened in Müller's fluid for three days showed a volumetric increase of 11.7 per cent. Other blocks hardened in Müller's fluid for three weeks, and then, transferred to alcohol and left in the latter for fourteen days, showed, on the other hand, a shrinkage in volume of 43 per cent. In alcohol, 95 per cent, at the end of twenty-four hours, a shrinkage in volume of 20.5 per cent was observed. This shrinkage is furthermore found to vary with different blocks from the same region of the same brain, and it is found that there is no constant relation between pieces from brains of various grades of hardening. Thus the method of fixation must always be taken into consideration. Again, in staining under exactly similar conditions, one section on a slide will be found more intensely stained than another section on the same slide, or even one part of a section will be found more intensely stained than some other part of the same section.

In the subsequent decolorization still greater variations may occur, for, if carried too far, important structures may be completely decolorized. If carried to the other extreme it is impossible to differentiate the various parts. The only way to avoid either extreme is by repeated examination of the sections under the microscope while the process is being carried on, as under similar circumstances one section in a given time may become much more decolorized than another. In regard to mounting of sections stained by the Nissl method, as all were that have been employed in this article, xylol-damar has given the greatest satisfaction to the writer. With this mounting medium there was no subsequent shrinkage, and thus a resulting exposure of the sections, also no decolorization was to be observed at later periods. In some sections mounted for over three years, no decororization was observed, even after several exposures to the intense light necessary for photomicrographic purposes, and frequent exposures to light in their examination under the microscope. Canada balsam was also used, but in time the sections were found partially or completely decolorized. Benzene-colophonium has a tendency to shrink and crack and thus expose portions of the sections in some cases.

It is desired to emphasize the importance of careful attention to all these details at the beginning of the article, in order to impress upon the reader the necessity of the greatest care and conservatism in the interpretation of any changes found in the specimens under observation, and, furthermore, the *correct* interpretation of the same. Before going on to the main subject of the article, a brief historical review of work in this line will be given.

*Historical.* The first study of the internal structure of the nerve cells began over fifty years ago when Remak (1841) called attention to a fibrillary structure inside the axis cylinder and cell-body of certain nerve cells. Later, Max Schultz (1872) studied nerve fibers and cells from various parts of the central nervous system of different animals with elaborate descriptions of his findings. He dealt with a description of the fibrillary appearance of the interior of the cell-body largely. This doctrine of the nerve cell was afterwards strongly supported by Boll (1873), Schwalbe (1885), and Ranvier (1878). Opposite views were, however, held by Arndt (1874), and Key and Retzius (1876). Arndt, in 1874, described the structure of the spinal ganglion cells and spoke of the presence in them of different kinds of elementary spherules which varied in size and general appearance. Key and Retzius stated that the ground substance of the spinal ganglion cells was homogeneous, but that in it numerous strongly refractive, round, oval granules were present, and they thought the appearance of a concentric striation or fibrillation could be simulated through the arrangement of the granules in rows. Flemming (1882) saw granules within the cells which would stain with nuclear and azo dyes and hematoxylin, but nevertheless affirmed a fibrillary structure of the central cells and of a tortuous or much curved threadwork within the spinal ganglion cells between the granules. In recently published articles (1896) he still maintains that fibrils exist inside the nerve cell protoplasm.

Dogiel (1893) has also expressed himself in favor of the views of a fibrillary structure for certain at least of the nerve cells. In 1885 Nissl published the first of a series of articles in which he calls particular attention, in tissues hardened in alcohol and stained in basic aniline such as majenta red and methylene blue, to certain structures. By Nissl's method these are brought especially well into view, and their arrangement in the protoplasm and their significance for the function of the cell can be studied.

These structures had been previously observed by Flemming and Benda (1882), but by less perfect methods. Nissl at first stained tissues hardened in alcohol with magenta red or methylene blue, and cleared in oil of origanum. This has been modified in several ways, so that at present his treatment is as follows: Small blocks of tissue are hardened in 96 per cent alcohol and fastened by Weigert's method with gum arabic, without imbedding. The sections are received in 96 per cent alcohol and stained in a watch glass. The stain is to be heated over the spirit flame until small bubbles arise which make a crackling noise (65° to 70° C.); sections are then transferred to aniline-oil-alcohol until differentiated. The process of differentiation is ended when no more coarse clouds of color go off into the fluid. The section is then transferred to the slide, dried with filter paper, after which some drops of oil of cajeput are applied, and the sections are again blotted with filter paper. A few drops of benzene are poured on the sections, blotted again, and finally some benzene-colophonium added, a cover glass placed over the section, and the slide heated until all the benzene gas has been driven off. Upon cooling, the section is thus permanently mounted.

The stain is made as follows: Methylene blue, B. pat., 3.75 grams; Venetian soap, 1.75 grams; distilled water or soft water, 1,000 c. c. The differentiating or decolorizing fluid has the following composition: Aniline oil, colorless, 10 parts; alcohol, 96 per cent, ninety parts. Nissl obtains his aniline oil directly from the factory at Hochst and keeps it carefully protected from the light. The benzene-colophonium is prepared by pouring benzene upon colophonium (white rosin) and alowing it to stand for from twenty-four to thirty hours. This results in a fluid transparent mass which can be brought to the desired thickness either by the addition of more benzene or by allowing it to evaporate, and then it is ready for use. In mounting, while driving off the benzene gas by heating over the flame, the material may catch fire, but if the flames are blown out immediately no injury is done, and the alterations produced by the burning are quite characteristic and easily recognizable. The above description has been given in detail as it is the classical method, which, as above and with various modifications to be described later, has been used by the writer in this article. This method distinguishes within the cell-body always two and sometimes three constituents. One of these constituents of the protoplasm stains entirely blue by this method, and is spoken of by Nissl as the stainable or visible formed part of the nerve cell. The second part remains entirely unstained, and is spoken of by him as the unstainable or the visible unformed part of the nerve cell-body. In addition to the above, in many nerve cells, pigmentary deposits are visible which have been especially studied recently by Rosin (1896). Nissl has prepared a somewhat elaborate classification of cells according to the character and arrangement of the stainable portion to the non-stainable portion of the cell-body in different cells in different parts of the central nervous system. This stainable substance shows a series of different forms; smaller and larger granules of regular or irregular shape, arranged in groups or rows. These stainable masses are sometimes arranged in threads, smooth or rough, and varying in thickness, course, and length. Often larger structures, regularly or irregularly shaped, are seen, which stain with varying degrees of intensity, some appearing homogeneous, others showing a complex internal construction difficult to describe.

Three varieties of these larger bodies are especially to be noted, (1) the so-called "nuclear caps" (Kernkappen), stainable masses in the form of regular or irregular cones hollowed out like a cap and corresponding to one pole of the nucleus upon which it rests. There may be two such caps within one cell-body at opposite poles, and occasionally, according to Nissl, three such caps may exist within a single cell. (2) So-called "wedges of division" (Verzweigungs-Kegeln), stainable masses completely filling the angle at the point of division of a nerve cell process.

(3) Spindles, oblong spindle-shaped stainable bodies, thick in the middle and thinner at both ends, the latter sometimes tapering off into thread-like filaments. These spindles may be one or two-sided. Any one of these forms may be vacuolated. Nissl makes a rather elaborate classification of these ganglion cells according to their internal morphology, after spending years in the most exact investigations of various nerve cells in the different nerve centers of man and animals. He has come to the conclusion that definite types or variations of nerve cells exist which are constant not only in the same animal but often exist characteristically in homologous localities in a whole series of animals. According to him, all the cells in the nerve centers, except the so-called chromophile nerve cells, can be divided into two main classes. The first class includes the nerve cells which possess a well marked cell-body surrounding the nucleus completely on all sides, the protoplasm having a distinct contour. These are called somatochrome nerve cells. The second class (sub-divided into two groups, cytochrome and caryochrome) includes those cells in which, in Nissl preparations, the nucleus is most in evidence, the nucleus has a clear contour, but only indications of a cell-body are present, an appearance due to either the scanty development of the cell-body or to the predominance in it of the unstainable substance.

These cells often look as if they were naked nuclei, though by Golgi's method it can be shown that they may possess definite axones and dendrites. In some of these cells the stainable substance may be present, but very unevenly distributed, at different points in the cell, the nucleus appearing as if only partially surrounded by protoplasm. Nissl classifies these cells with such an ill-defined cell-body, the nucleus appearing only partially surrounded and not exceeding in size the nucleus of a neuroglia cell or of an ordinary leucocyte, as "granules" (Körner) or cytochrome nerve cells. These cells are present in great numbers in the granular layer of the cerebellum, also in the cerebral cortex and the olfactory bulb—being different varieties and not identical. The second subgroup of cells, in which the cell-body is only indicated but the nucleus is of the size of an ordinary nerve cell, or at least larger than that of a neuroglia cell, he designates as caryochrome nerve cells. There are varieties of these as in the substantia gelatinosa Rolando, and those of the ganglion habenula—these types being designated at present by letters of the Greek alphabet. A majority of the nerve cells fall in the first group—the somatochrome cells—where the cell-body, in the light of morphology, has apparently greater relative importance than the nucleus. In this group are a series of types of nerve cells to be distinguished from one another partly through differences in the nuclei, but chiefly through different relations of the stainable and unstainable constituents of the cell-body.

These somatochrome cells were originally divided by Nissl into four groups, but now he includes them all in three groups as follows:

Group I. Arkyochrome nerve cells—stainable portions of cell-body in Nissl preparations in the form of a network, branches of which appear to be distinctly connected. Nissl notes, however, that in many of these cells processes can be made out into which the distinct network of the perinuclear part of the cell-body can go over, thus forming a parallel-striped arrangement. These arkyochrome cells Nissl subdivides into enarkyochrome forms and ampharkyochrome forms. The former shows the stained constituents arranged in the form of a network, which differs from the latter in that the intensely stained radiating nodal points of the network are connected in the cell-body by deeply stained thick bridges so that a further connected network of very deeply stainable substances is observable. Both these sub-groups of cells are widely distributed throughout the central nervous system. The former have been observed by Nissl in the spinal cord, but are most numerous in the large dorsal nucleus at the proximal end of the medulla. What he formerly classified as Group III, arkyostichochrome nerve cells, are now included in this first group (1897a). He describes these cells as presenting a striated appearance with a network-like structure united in a most intricate manner.

The Purkinje cells of the cerebellar cortex were given as typical examples of cells of this sort. (See Plate IX, Fig. 27).

Group II. Stichochrome nerve cells, stainable substance arranged in striæ, running in same direction, and usually parallel to contour of cell-body, in part also with surface of nucleus. These striæ, made up of different stained elements, threads, spindles and granules, are more or less isolated and in rows. Nissl describes four types of these cells represented by (1) nerve cells ot motor nuclei, (2) large cells of cornu Ammonis, (3) certain cells of cerebral cortex, and (4) certain cells of spinal ganglia.

Group III. Gryochrome nerve cells, stainable substance entirely made up of small granules distributed in threads or heaps. Nissl does not give an illustration of these cells, but states that they are to be found particularly but not exclusively in the corpus striatum. Nissl states further that transitional forms exist which are difficult to definitely classify, but that the cells of a wholly definite structure are situated throughout the animal series in homologous localities. He has also shown that all these types of cells may, under certain circumstances, show different staining relations (1894a), that is, the individual members of a certain group of cells belonging to one type may be palely, moderately, or intensely stained. This difference seems to depend upon the concentration of the stainable substance in the cell-body. The darkly stained cells he designates as pyknomorphous cells, in which stainable portions are arranged relatively most closely; intermediate stages are designated as parapyknomorphous cells; while very feebly stained cells are designated as apyknomorphous, in which stainable masses are not arranged close to one another but are somewhat widely separated by the non-stainable constituents of the cell-body. Nissl furthermore mentions that often the nucleus shows modifications which correspond in greater or less degree to the staining intensity of the cell-body, so that in an apyknomorphous cell the unstained nuclear substance is relatively more abundant than in the pyknomorphous cells. This holds in the somatochrome cells, and to a less degree in the caryochrome and cytochrome cells.

Supposed to be more or less in the nature of an artefact are the so-called chromophile nerve cells which one finds often single or in small groups along with the other nerve cells, but in which the stainable substance seems to be evenly diffused throughout the cell-body, making it impossible to distinguish a stainable from an unstainable constituent in the cell. Nissl (1896c) states that they are always relatively smaller than pyknomorphous cells. The explanation of these forms is not as yet entirely satisfactory, but they are supposed to be due to the action of the reagents employed, though under certain circumstances they may have a pathological significance. Another nomenclature has been introduced by Flesch (1897), in which he speaks of chromophilic cells and chromophobic cells with transitional forms, and attributes the differences to variations in the internal chemistry of the cells, this latter depending in part upon differences in the devolopment, in part upon differences in metabolism or function.

Von Lenhossek (1895) gives a very accurate description of the appearances within the cells of the ventral horn of the spinal cord and in the cells of the spinal ganglia. He has found that thionin stains as well if not better than methylene blue, and Barker (1899) has obtained similar results, but states that crystalline deposits have been more frequent in preparations stained with thionin than in those stained with methylene blue. In material hardened in Lang's solution or other corrosive sublimate solutions, the writer has at times detecfed crystalline deposits, whether stained with thionin, methylene blue or methylene violet, and attributed the same to the fixative fluid and not to the stain at all, as no crystalline deposits have been observed with other fixative agents—alcohol, formalin, and Van Gehuchten's fluid—regardless of which stain was used.

It would be interesting to know whether corrosive sublimate fixation was used by Barker in the above mentioned preparations. Van Gieson uses polychrome to a considerable extent as a stain, but it seems to the writer to give a paler and less distinct stain than either methylene blue or methylene violet, and has a tendency to fade after a time. Referring to von Lenhossek again, he objects to the term "granules" for the stainable substance, the masses being ordinarily much too coarse to be so designated. He has given accurate descriptions of their characteristics and arrangement in various animal species. This stainable substance of Nissl he designates as "chromophile corpuscles," and later uses the term "tigroid masses" (1896).

De Quervain (1893) has suggested that the chromophilic bodies or corpuscles are aggregations of fine granules, but von Lenhossek refuses to admit that all such bodies represent aggregations of minute granules. Schaffer (1893) first described the peculiar behavior of the axone and adjacent portion of the cell-body known as the axone hillock as regards Nissl staining, this space being entirely free from this stainable substance of Nissl. With Kronthal's method the axone and axone hillock stain intensely in methylene blue, but Benda (1895) found that when specimens thus prepared were cleared in creosote this region lost its color and only the stainable substance of Nissl in the cell-body and dentrites retained its color. He found one exception, however. In the basal axones of the pyramidal cells of the cerebrum, especially of those known as the giant pyramidal cells of Betz, just at the beginning where a collateral is coming off at right angles, a small wedge-shaped granule, triangular in section, takes up the methylene blue, the axone itself not staining at all. These different results obtained by differences in technique emphasize the writer's previous statement of the necessity of a full knowledge of the history of the case and every detail of technique before coming to any conclusions in a given case.

With this brief review of the more important literature on the internal structure of the nerve cell we will now turn to the arrangement of the cells in the cortex. To go into the literature of this subject in detail would be sufficient material for an article in itself, so that it will be passed over as briefly as possible. Table I shows the arrangement of the cells as described by the more important contributors to this subject. As early as 1782 Gennari divided the cortex at the posterior pole of the cerebrum into three layers, an external and internal gray layer separated by a white layer (so-called line or band), the "line of Gennari." Vicq d'Azyr, in 1786, described this region as also made up of an inner and outer gray layer between which was a white layer (line or band), the "line of Vicq d'Azyr." Meckel, in 1812, stated that the gray layer of the cortex is single in all parts but the occipital region and cornu Ammonis. In the occipital region a band of white substance, the line of Vicq d'Azyr, separates the gray substance into an external and internal gray layer, so that the structure of this part of the brain is more complex than the rest of the external cortical region. Coming down to 1840 Baillarger first examined the cortex carefully, and described three gray and three white layers, or six layers in all, alternating gray and white. From his descriptions the line of Gennari or Vicq d'Azyr, in the occipital region, received the additional name of the "line of Baillarger." In 1841 Remak examined the cortex microscopically as well as macroscopically, but without the use of staining agents, and described a four-layered type made up of two gray and two white layers alternating with one another.

Somewhat later (1852) Kölliker described a three-layered plan, the inner layer appearing of a yellowish red color, the middle of a grayish color, and the outer of a whitish color.

Berlin, in 1858, first used stains in his work, hardening pieces of the cortex in chrome salt and staining with carmine. He described six layers as the type, the upper five from the character and arrangement of the cells and the lower as a layer free from cells. He describes the layers from within outwards toward the surface, reversing the usual order of all other authors, and leading to considerable confusion, unless this fact is carefully noted. Clarke, in 1863, described eight layers as the common type, and Luys the following year, 1864, described just one half that number, or four layers, as the common type. Arndt, in 1867, described a five-layered type, and Meynert in the same year divided the cortex into a motor and a sensory region, the former anterior to the fissure of Rolando, and the latter posterior to the same. He described five layers as the common type, and eight layers as the type in the occipital region. Cleland, in 1870, described rather a complex arrangement of the cortex into a four-layered type, the third layer being separated from the fourth by what he designated as the "primary pale band." The "deep pale band" is included in and forms the lower part of the fourth layer. Henle in the same year described a four-layered type made up of (1) the outer layer, (2) outer spherical cell layer, (3) pyramidal cell layer, and (4) inner spherical cell layer. Charcot, the following year, 1871, described the layers of the cortex as five in number, (1) superficial layer, (2) small pyramidal cell layer, (3) larger pyramidal cell layer, (4) granular cell layer, and (5) fusiform cell layer; approximating that used by many of the subsequent authors. Lewis, in 1876, described five layers as the type for the motor area and six layers as the type for the sensory area. Major in the same year, 1876, described six layers as the common type, and Krause, also in the same year, described seven layers as the common type. Betz, in 1881, made the division into five layers as the type for the motor area, but divided the sensory region into eight layers as the type—a classification somewhat similar to Meynert's, but with a different nomenclature.

Golgi, in 1883, using his impregnation method, divided the cortex into three layers as the type, (1) superficial or small pyramidal cell layer, (2) middle or larger pyramidal cell layer, and (3) internal or irregular cell layer. Schwalbe, in 1885, described a curious arrangement of the cortex into four layers, the upper two known as the "outer Hauptzone" or chief zone, and separated from the lower into two layers, known as the "inner Hauptzone" or chief zone by the "boundary zone" or "line of Baillarger." Obersteiner's (1887) division into a five-layered type is similar, except as to the nomenclature, to that of Charcot's common type, Lewis' motor type, and Betz's motor type. Cajal, in 1890, recognized the same three layers as Golgi in the common type, but added a fourth layer, which he called the first or molecular zone, then followed the second, or zone of small pyramidal cells; the third, or zone of large pyramidal cells, and the fourth, or zone of polymorphous cells; thus making four layers in all. Golgi's first layer is thus seen to include the first and second layers of Cajal. In the occipital lobe, Cajal describes an additional layer of vertical fusiform cells between the first or molecular layer and the layer of small pyramidal cells, the other layers being similar to his common type, and thus making five layers in this region. Gowers, in 1893, adopted practically the same division into a five-layered motor type and six-layered sensory type as described by Bevan Lewis in 1876. Hammarberg, in 1895, described six layers as the type, with numerous variations in different parts of the cortex. Edinger, in 1896, describes four layers, similar to Cajal as the type, and Starr (1896), in his atlas of nerve cells, also describes a similar arrangement. Nissl (1898) describes a four-layered type as follows: First, or layer poor in cells; second, or layer of pyramidal cells containing 2 equals layer of small pyramidal cells (equals 2 of Meynert's layers) plus 3 equals layer of larger pyramidal cells (equals 3 of Meynert's layers); third, or layer of small cells (equals 4 of Meynert's layers); and fourth, or internal (6) plus external (5) zone of the layer of medullated fibers (equals 5 of Meynert's layers). Region 5 equals ganglion cell layer, and region 6 equals spindle cell layer of Hammarburg. It will be seen that there is much variation and not a little confusion in adjusting these various arrangements and classifications to one another, and in order to simplify the matter as much as possible the writer suggests that as the large and small pyramidal cells are so intermingled and merged into one another, they should be included in one layer, thus making three layers the common type, as follows: First, or superficial layer, corresponding to the first layer of Cajal and Edinger; second, or pyramidal cell layer, corresponding to the second and third layers of Cajal and Edinger; and third, or spindle cell layer, similar to the fourth layer of Cajal and Edinger. This arrangement differs from Golgi's three-layered type in that the latter recognized no superficial or tangential fiber layer, but includes this as the upper part of his first, or layer of small pyramidal cells, whereas in the writer's arrangement one pyramidal cell layer includes both the large and small pyramidal cell layers of Golgi. The third layer of Golgi and of the writer are the same as the fourth layer of Cajal and Edinger. This classification into three layers as the type will be used for convenience in the subsequent description of the plates. That there are variations from the type in different parts of the cortex, of course, is recognized by all who have worked in this field to any extent, and, furthermore, specialized parts of the cortex have a specialized arrangement, as the cornu Ammonis, for example.

In turning now to the pathological investigations by means of the Nissl stain (1898), they are found to have been exhaustive and varied, including most parts of the central nervous system in both man and animals and in many forms of disease. A brief review only will be given of the contributions containing descriptions of researches by Nissl's stain in that form of mental derangement known as dementia paralytica, or general paresis. Nagy (1894) carried on his investigations by means of the Nissl stain, and found the greatest changes in dementia paralytica, including various stages of cell degeneration up to complete destruction of the same—the cells most altered being those of the frontal lobes, and least changed those of the occipital lobes. Changes of a high grade were also shown in the cells of cases dying after severe epileptic insanity—here the gyrus rectus and cornu Ammonis being affected the most. In chronic forms of insanity similar changes were found, but the number of cells entirely destroyed was undoubtedly smaller than in the above mentioned forms of illness. In acute hallucinatory confusional insanity only the beginning stages of alteration were found; similarly in mania. He finally states that each form of mental derangement showed the highest grade of change in which the clinical picture of the worst suffering was present, while in the curable forms there were found corresponding slight changes. Belmondo (1896) employed the Nissl method in investigating the alterations in the nerve cells in dementia paralytica, and did not find changes of great gravity—at most the cell protoplasm being much diminished and disintegrated; now and then pigmentary atrophy, as much in the Rolandic region as the frontal lobe, is found, and in other parts a diffuse chromatolysis is to be seen. He condemns the expression meningo-peri-encephalitis, which implies a conception of an inflammatory process.

Boedecker and Juliusburger (1897) examined sections of cortex from the central and parietal convolutions of three cases of dementia paralytica by the Nissl method. They found thickening of the pia with septa projecting into the cortex, containing blood vessels whose walls showed no special thickening but were surrounded by rich deposits of pigment granules, which latter were also found here and there, distant from the septa in the cortical network. In regions most affected the cortex did not present its well known layering. There was to be seen a thickly-compressed granular crowding with different shaped granules; thin, spindle-shaped, round, or oval, with strongly colored ones lying adjacent to those only slightly colored. There was increase of blood vessels and hypertrophy of the interstitial network, with corresponding decrease in the number and size of the cells. Many cells were considerably diminished in size, markedly shrunken, entirely without processes, and intensely colored. At times a differentiation into nucleus and granular cell-body was not possible, owing to complete chromatolysis, and very seldom were cells found with a strongly colored nucleus, strongly colored fine granules about the same, with larger granules at the periphery of the cell-body—a partial chromatolysis.

By the Marchi method these investigators determined a degeneration of the fibers from these cells extending into the spinal cord. They conclude the process to be an intense degeneration and proliferating one, going on hand in hand—degeneration of the cells and proliferation of the interstitial network, with increase of blood vessels. Crisafulli (1897) notes a great variety of changes in dementia paralytica. The cellular changes are most advanced and diffuse in the frontal lobes, but are not limited to that region. He examined sections from the frontal, parietal, occipital and temporo-sphenoidal lobes of both hemispheres. He found pallor, granular disintegration, and loss of chromatic substance. Often the cell-bodies were atrophied or contained an excess of yellowish pigment, and their numbers were reduced. The nuclei were often eccentric, and all stages of the destruction of the nucleus were observed. While the alterations shown by Nissl's method were not less constant than those demonstratable by other methods; Crisafulli does not consider them characteristic of the disease or in any way different from those seen in some other mental diseases. They are, however, more or less grave and diffuse and not limited to a single cortical center; with varying degrees of degeneration of the nerve cells. He further states that, provided the nerve cell element degeneration is not greatly advanced, it is impossible to find any alteration of the blood vessels, and, finally, that when the psychosomatic condition of the paralytic is not greatly aggravated and death intervenes from other causes; it is possible that the pathological report will note some elements which are not degenerated, although there may be various alterations. His article is illustrated by eight figures showing cells in various conditions of degeneration.

Angladi (1898) reports a case of acute dementia paralytica in which death occurred after a series of epileptiform convulsions when the patient was at the age of thirty-seven years. The autopsy showed peri-encephalitis. Preparations were made from the ascending frontal convolutions and the anterior part of the frontal lobe of the left hemisphere. He states that not a single one of the pyramidal cells preserved its normal characteristics, and the transformation in the great majority of cases semed to be various stages of the same process—various stages of chromatolysis, vacuolization, eccentricity of the nucleus up to rupture of the cell wall and extrusion of the nucleus and, finally, complete destruction and disappearance of the cell. The chromatic substance is first attacked, and some few cells relatively healthy showed about the nucleus the first stages of dissolution. Destruction of the achromatic network was shown by formation of vacuoles at the periphery of the cell, increasing gradually up to complete destruction of the substance of the cell. The nucleolus becomes vacuolated and disappears. The nucleus is attacked by central chromatolysis and disappears *in situ,* or becomes eccentric, or the cell-body may rupture and the nucleus be extruded, becoming irregular, compressed or shriveled. The contour of the cells is always irregular, and the prolongations either broken or tortuous. He states that the cells of the medulla and cord show identical alterations. Angladi finally concludes by stating that we do not know whether these lesions are the cause or the result of the malady, and asks the question, "Are the lesions primary or secondary to an alteration of the vessels or interstitial tissue?"

Berger (1898) examined the anterior horn cells of the spinal cord in twelve cases of dementia paralytica and found lesions affecting principally the chromatic substance in 83 per cent of the cases. He failed to find a strict parallel between these cellular lesions and those of the fibers and cortex, or between them and the clinical symptoms of the disease. He illustrates his article by figures of these cells.

Nissl (96a), in 1896, stated that he maintains the same position as Kraepelin, namely, that one sees in dementia paralytica a general disease with the histopathology directed especially to the cortex. The pathological changes in the blood vessels are obscure, and the relation of the glia to the blood vessels is complicated, also the condition of the lymphatic vessels. Some authors regard the disease as an inflammatory process, others that a chronic interstitial inflammation enters into it, others that it is a parenchymatous process, and still others that it is a histopathological process in which the specific tissue is diseased primarily. Nissl notes that an inflammation without the blood vessels sharing in it cannot be thought of. He states that the appearance of the paralytic cortical disease can be present without the blood vessels being diseased and without the blood vessels containing any elements of an inflammatory process. Also there may be a high grade of disease of the tissue (Gewebe) in excess of any disease of the blood vessels, and only a slight involvement of the tissue, and *vice versa,* a severe tissue damage with only insignificant disease of the blood vessels. Also direct inflammatory blood vessel changes with infiltration of the walls of the same with leucocytes and "mastzellen," which may rarely pass out into the adjacent regions. In some cases this inflammatory alteration of blood vessels results in a massive production of decay of numerous neurones. These inflammatory changes in the blood vessels have nothing directly to do with the chief paralytic process, and are only found in the cortex when sepsis-producing bacteria are present. It therefore follows that the paralytic cortical disease can be regarded either as the result of disease of the blood vessels or as an inflammatory process. Almost all cases of dementia paralytica show a slight or severe disease of the blood vessels. There may be also a leptomeningitis, disease of the beginning part of the aorta, an injury to the diplœ, brittleness of the bones, or there may be a general arterio-sclerosis. If it is not a disease of the arteries or an inflammatory process, it may be a primary disease of the glia or of the cortical neurone. Changes in glia are progressive in kind (mitosis of glia nuclei, hypertrophy of glia cells, and increase of glia fibers) with regressive changes in the cortical neurone—an acceptation that results of investigation directly contradict, since we find in the most luxuriant increase of glia that the nerve cells are only slightly or not at all changed, and *vice versa,* in the most severe nerve cell changes the increase of glia may be only slight. The acceptation of a primary glia fiber increase is absurd, since the glia fibers are an intercellular substance. Nissl therefore concludes that the cortical disease of dementia paralytica is a primary disease of the cortical neurone; at the same time with the regressive changes in the cortical neurone goes the progressive changes in the glia cells. Histopathological investigations of paralytic cortical disease has to deal chiefly with the following difficulties: (1) a paralytic diseased cortex and the, to us, available cortex of the dead paralytic are two different things. Entire series of original circumstances that damage the neurones of the cortex without having anything to do directly with the paralytic process is to be noted, as the complete closure of a blood vessel which only indirectly bears upon the original process. Also death of a paretic from typhus fever, septic pyemia, etc. The problem is extremely difficult, as there is no specific disease of the cortical cells, and, furthermore, there is no paralytic cortical cell disease, although there is a paralytic cortical disease. The kind of disease of the cortical tissue is worthy of being pointed out. (2) It is not sufficient to know that the cortical neurone is diseased, but it is important to know which cortical neurones are damaged. Attention to the kinds of nerve cells directly diseased is important for the real conception of the disease process, and also for the critical examination of the plan of the cortex and its functions. Nissl differentiates the following forms of disease of the cells of the cortex in dementia paralytica: (1) Acute progressive disease. In certain cases the disease ends with the complete destruction of the elements of the cortex. (2) The chronic disease progresses slowly, resulting in either a pigmentary degeneration or in a decay of the cell-body and nucleus, and ends with so-called cell sclerosis. (3) The severe disease of the cortex which runs either an acute or subacute course and terminates with the death of the cell. The necrotic cells persist commonly either bleached out or having the appearance of colliquation or of vaucolization of the ground work. (4) The combined form of the disease, in which the cell may be acutely diseased without either a cure following, or, on the other hand, the ordinary course being taken that terminates with the destruction of the cell. Midway in its course the disease process is arrested and takes on the symptoms of the chronic disease. The severe cortical cell disease is entirely overlooked by former authors. It differs in that the nucleus is also involved and a process of liquefaction takes place in the same. It becomes smaller, shrunken, the contours become homogeneous and tinged, the nucleolus sinks to the nuclear wall, which latter is irregular and shriveled into folds, the network cannot be distinguished, and vacuoles and crystals may be formed. In the necrotic cells calcareous deposits may be found, as is seen in other cortical cell diseases, as sclerotic elements. These chalky or calcareous concretions occur in the form of fine granules, crumbs, placques, or stalactitic masses, which are intensely colored with methylene blue. The entire cell may be bleached or only a single part, as some of the fine dendrites or only a single dendritic process, or only the nucleus, or, finally, only the nucleur membrane. This calcification, moreover, is an exceedingly important phenomena, since we are entitled to conclude that partially calcified cells are no longer functionally active and are necrotic. Under similar conditions in acute diseases we recognize the phenomenon of death in the affected cells. If one places the preparations in alcohol after twelve hours postmortem, any mistake in this direction may be obviated. Whoever grasps the histopathology of the cortical cells will guard against any mistake in regard to the above mentioned death phenomena. Ewing, in 1898, used the Nissl method in observations upon the changes found in ganglion cells of the central nervous system in various pathological conditions, and stated that various grades of chromatolysis were found in the cortical cells in dementia paralytica.

Ballet (1898) exhibited sections from the paracentral lobule of both a normal and a paretic brain, stained by the Nissl method.

In the normal brain, under a magnification of forty-five diameters, the four layers of Schwalbe and Ramon y Cajal were easily discoverable, but in the paretic sections, in marked contrast, they were recognized with difficulty; also in the latter, great numbers of capillaries were noted in the third layer and white subcortical substance, the nerve cells were less numerous, and in all cases less distinct and lost in the midst of a mass of nuclei. At 130 diameters the contrast was still more marked, and at 250 diameters vascular lesions were noted consisting of enormous dilatation of the capillaries and arterioles, the investment of these vessels by a casing of lymphatic corpuscles which distended the adventitious sheaths, and the accumulation of pigment at certain points, particularly in the neighborhood of the bifurcations. These alterations are also revealed by hematoxylin and picro-carmine, and have been described for a long time by all observers, but the Nissl method shows the changes more clearly than any of the others.

In addition to these changes one may mention the multiplication of the white corpuscles and their migration from the vessels by diapedesis, as due in part perhaps to the proliferation of cellular elements of the neuroglia which accumulate in the interstitial tissue, principally in the neighborhood of the vessels or about the nerve cells. At 600 diameters one easily distinguishes the small white cells (lymphocytes), with small nuclei deeply colored and with but a small amount of protoplasm; large white globules with protoplasm somewhat abundant and with voluminous nuclei irregular in form and less impregnated than those of the lymphocytes by the methylene blue; and, finally, polynuclear leucocytes. But interest in Nissl's method is chiefly in the study of the lesions of the nerve cells. At 600 diameters one sees the profound alterations undergone by these cells. In examining the elements of the third layer (large pyramidal cells) or the giant cells of Betz, there is a tendency in them to lose their triangular shape and become oval or rounded, the protoplasmic prolongations are atrophied and but slightly visible, the chromophilic granules for the most part undergo a process of disintegration and are reduced to a sort of fine powder or dust, or are entirely dissolved in the mass of protoplasm. The author then discusses the nature of the process, some authors (Magnan, Mierzijewski, Mendel) claiming the primary disease to be that of the neuroglia framework—an interstitial encephalitis. Others on the contrary (Tuezeck, Ziegler, Binswanger, Joffroy, Pierret) claim it affects primarily the nervous tissue, either the nerve fibers or the nerve cells; thus being a parenchymatous encephalitis. The more Ballet studies the pathological anatomy of dementia paralytica the more he is convinced that the first and most important lesions are those of the blood vessels. These are seen in every case, while those of the cells are inconstant, variable in degree, and subordinated to those of the vessels. This is not to say that the cellular alterations may be, as some authors claim, the result of the mechanical choking of the cells by the proliferating interstitial tissue which makes their pathology more complex, but he thinks the result is due less to the compression by the thickened neuroglia than to the difficulties of assimilation, owing to either the circulatory obstruction or the action of the toxines carried by the blood. Ballet then discusses the relation of syphilis to dementia paralytica, and states that the pathological anatomical findings in the two cases are practically identical, and advocates a syphilitic etiology for the disease.

In this historical review of the literature on the pathological conditions found in dementia paralytica by the employment of the Nissl method, it is to be noted that but few of the contributions were accompanied by figures illustrating the various pathological findings described in the text.

*(To be continued.)*

# CASES OF COMBINED MORPHINE AND ATROPINE POISONING.[1]

By Philip Zenner, A.M., M.D.,

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These cases were deemed worthy of report because none like them, so far as I could learn, have been published. They seem to indicate that poisoning with the two drugs combined is especially dangerous, notwithstanding their supposed antagonism.

The first case was reported at the time of its occurrence some years ago. The patient was a nurse, of twenty years, who had taken with suicidal intent, hypodermic tablets each containing one quarter of a grain morphine, and 1-150 grain atropine. Nothing was known of this at the time, and it was only discovered hours afterwards when, after a careful search, some tablets were found under her pillow. It was supposed, judging from the number of tablets remaining in the bottle wherein they were contained, that she had taken about eight of them, but that may have been far from the mark.

She was found to be unconscious at 6 P. M. I saw her a few hours later. At that time she was profoundly unconscious. There was complete flaccid paralysis of all the extremities. The face was expressionless, eyes slightly divergent, the pupils of medium size and irresponsive to light. Pulse was 120 per minute, of fair force and volume. Temperature 100. Respiration eleven per minute, and very irregular. Marked cyanosis, knee-jerks could not be elicited. Urine contained no albumin, specific gravity 1,012.

All efforts at resuscitation were futile—washing out the stomach, introduction of coffee through the stomach tube, hypodermic injections of whiskey and ether, applications of the electric brush, etc. The breathing was the ominous feature throughout, quiet, never stertorous, but irregular, the interval between the respirations becoming gradually longer and longer. The pulse, which was good, though rapid, throughout, could be felt for some time after the breathing had ceased. The pupils gradually became more dilated, though never to the degree of full dilatation. She died at 3.30 A. M.

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[1] Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

The second case was a young girl of thirteen years, who had chorea for about two weeks. Her restlessness was so great that the physician left for her some hypodermic tablets, each containing morphine gr. one-eighth, and atropine gr. 1-200. She received of these tablets one at 8 P. M., one at 9 P. M., one at 10 P. M., one at 11 P. M., one at 11.30 P. M., and one at 12 P. M.; that is, six tablets within four hours. She fell asleep an hour or two after the last tablet had been taken, and slept soundly until 6 A. M., since which time she was dozing and waking, and when awake delirious, until I saw her. I made a hurried call about 11.30 A. M., the only time I ever saw her. She seemed asleep when I stepped into the room, but soon awoke, and was quite restless. At times she would squirm about and scream, "Oh, that cramp," doubtless on account of abdominal pain. She gave sufficient answers to questions to indicate that she was fairly conscious, those in attendance stating that she was now more lucid than she had been at any time during the day. Her temperature was 104° F. Pulse was rapid and thready. Scarcely to be counted, but about 130. She was very cyanotic. Her heart could not be carefully examined, and her restlessness prevented a satisfactory examination of reflexes. She had vomited everything she had taken that day.

As nearly twelve hours had passed since taking the last of the atrophine and morphine and her mind was becoming more lucid, I thought the prospects for recovery fair, even though her condition appeared critical. Nevertheless I telephoned at once to her physician, telling him she ought to be stimulated and carefully watched. A few hours later he said her pulse was fair, and he thought she was doing well. He did not see her again. Perhaps she was doing well, but about 8 P. M. she was seized with severe convulsions and died within an hour. I have little definite information of her condition subsequent to the time of my visit.

I saw so little of this second case that I can scarcely make a comparison of the two cases. The most striking difference was in the pulse, which was very good in the one and bad in the other. That may have been due to the fact that the proportion of atropine was much larger in the latter.

I know of but two reported cases of a somewhat similar character, and these I mentioned in my former report.

The first was that of a man of sixty, who took by mistake a tablespoonful of a mixture containing equal parts of liniment of belladonna and tincture of opium. Two and one half hours after swallowing the mixture he was unconscious, pulse 140, breathing rapid, pupils dilated and sluggish. Twenty-four hours later he appeared to be out of danger. Four hours subsequently he sat up in bed to take a drink and fell back dead.

The second case was a woman of seventy-nine, who took by mistake ten grains extract belladonna, ten grains of extract of conium, and eighty minims of tincture of opium. Two hours later she lay in profound coma, breathing hurried and noisy, pupils dilated, pulse 130, feeble and intermittent. The patient was resuscitated to the extent that she took nourishment fairly well, but she died forty-eight hours subsequently of exhaustion.

These two cases are, perhaps, not to be compared with my own, as age and enfeeblement may have led to the fatal termination. But nevertheless so far as they go, they seem to lend support to the view already expressed, that poisoning with a combination of the two drugs is peculiarly dangerous.

# A NOTE ON PERIODIC INSANITIES WITH REPORT OF THREE CASES OF INTERMITTENT MELANCHOLIA.[1]

By Alfred Gordon, M.D.,

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The periodicity of certain forms of insanity has not been frequently the subject of a special study by the alienists. The reason of it lies probably in the fact that cases of this nature are rarely met with in institutions. Yet, outside of asylums, they are not as rare as it may appear.

The writer has had the opportunity to have under his personal observation quite a number of cases in and outside of asylums. A close study of them leads him to consider the subject of sufficient importance from both the scientific and practical points of view, so that he considers himself warranted to report the result of his observations.

Under the term Periodic Psychoses we understand attacks of mental derangement occurring at certain intervals, during which the patient is more or less free from mental disturbances. Some alienists, especially the older ones, maintain that the intervals between the attacks must be of equal duration in order that the psychosis be called periodic insanity. The reading of the records, however, shows that the duration of the intervals and of the attacks of the psychoses are not at all characteristic features of periodic insanities. Their duration varies.

The periodic psychoses known are mania, melancholia, and paranoia. Krafft-Ebing and Kraepelin also include the circular insanities.

While mania is more likely to occur periodically than the other forms, melancholia is by no means infrequent. The reason why the periodicity is often overlooked is that it is not easy to keep this class of patients under observation a sufficient length of time.

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[1] Read before the Philadelphia Neurological Society, April 28, 1903.

Among all the cases that came under my observation, there are three that I have had the good fortune to have under my care for the last four years, and thus have had an unusually good opportunity to follow the individual attacks step by step and keep complete records. A close study of them showed me the remarkable uniformity in the character of the onset of the attacks, in the character of the termination of the attacks, in the age, in the family history, and in the fact that each subsequent attack was more and more pronounced. A still closer study will make us see clearly that in patients presenting the periodic forms of insanities, there is undoubtedly an unusually pronounced degenerative basis, upon which are built these occasional outbreaks of depression with or without delusions. The histories of the cases are as follows:

Mrs. E. O., aged thirty-five, was seven times pregnant. Four years ago, after her fourth confinement, in the midst of apparently perfect health, she became suddenly depressed and felt she could not do her work. As soon as she commenced to work she imagined she could not continue because she feared not to be able to do it properly. Soon her worriment increased, because she could not do anything for her children, whom she loved very dearly. She used to prepare the food for them, and now she found herself incapable of doing it. Conscious of this condition, the patient soon reached a state of complete discouragement. She ceased to take an interest in her household, in her husband and children. The painful moral depression became associated with the idea of physical and moral impotence which showed itself in the lack of confidence in herself and in the absence of initiative and determination. The patient's life was dominated by sad ideas; she could not take pleasure in anything; everything and everybody became to her a source of constant irritation and suffering. Tortured by the complete inability to accomplish anything and to render the slightest service to her family, to whom she was previously a devoted mother and wife, she could not understand why she suffered so much. She then arrived at the conclusion that she was a worthless being condemned to perish. At the same time the struggle between the state of consciousness of this miserable existence and the inability to accomplish anything put the patient in a state of constant anxiety which had its effect also upon her vegetative life. She lost totally her appetite and would never ask for food; her digestion suffered considerably. The special senses became also affected; the taste and smell were so perverted that she rejected almost every article of food with disgust. It was astonishing to hear the husband telling how small was the quantity of food the patient received: Only two or three times a day she would take a spoonful of milk and a couple of slices of bread. She soon began to lose in weight. The respiration was slow and superficial. The pulse was small, filiform and accelerated. Pain in the precordial region was frequent. The blood examination at different periods showed a condition similar to that of chlorosis with the exception of a marked leucocytosis (22.000 per ccm.).

If we add to these symptoms headache, partial insomnia, with frightful dreams and occasional visual hallucinations, the picture of the patient's condition will be complete.

The mental state just described remained almost unchanged for two months. Only now and then a ray of hope would make its appearance. She would cease to groan and moan, complain less and speak less of self condemnation; then the somatic symptoms would become ameliorated. These slight remissions would last only a few hours.

At the expiration of two months, one morning the patient awoke in a calm and peaceful manner, called her children, began to caress them and offered to prepare breakfast for the family. The mental faculties became clear and normal. She began to take an interest in her surroundings, in her own personality; briefly speaking, the melancholic state by which she was tortured for two whole months disappeared entirely.

She became pregnant again and felt well during ten months. Three weeks after the confinement, which was normal, suddenly, and without any preliminary symptom, the old mental trouble returned. Again the same complaint of worthlessness, the same indifference to her surroundings, again the same state of anxiety which made her this time speak of suicide. The knowledge of the fact that this was her second attack made her fear that her case was lost, that she would never get well, and therefore the symptoms of anxiety and the somatic symptoms were more pronounced. The visual, also auditory, hallucinations occurred very frequently. This state of depression lasted two months. Exactly like in the first attack, this second one also terminated abruptly, and again she became normal. A third pregnancy occurred. Six weeks after the confinement the old trouble returned. Without any prodromal symptom she became markedly depressed. The self condemnation with the delusions of worthlessness, also the tendency of suicide, hallucinations and the painful emotional state recurred.

Shortly speaking, the patient passed through a state of mental disturbances almost identical in its evolution with the first two attacks, with this difference, however, that the last attack lasted four months, while the first two attacks lasted only two months. The termination was also abrupt.

The patient is now perfectly well. A year elapsed since the last attack. She is not pregnant.

In her family history I could elicit two cases of insanity—in a sister and grandmother. She had eight children, two of whom died with hydrocephalus, one died immediately after birth; one of diphtheria.

Her personal previous history shows that she suffered from obsessions at the age of sixteen, and was always considered highly irritable.

Case 2. Mrs. B. B., aged thirty-one, came under my observation three years ago with a typical melancholia. Delusions of unpardonable sin of a religious character, hallucinations, both visual and aural, and an array of neurasthenoid symptoms. All these symptoms were present at the time I first saw her. From the previous history of the case I could detect one previous attack, which was evidently mild in character, but was undoubtedly present. The second attack, in which she came under my care, was sudden, without a preceding physical indisposition or any mental disturbance. As a woman of means, she enjoyed life, and was mentally lucid. One morning, after having spent the previous night in a theater, she awoke in a state of depression, and from that hour on the above-mentioned symptoms gradually developed.

A peculiar fact to note is that this attack, as well as the first, which I did not witness, began with a facial neuralgia which lasted only one day.

The condition lasted three months. The termination of the disease was strikingly abrupt. Six months later, apparently without any cause, the patient again became depressed. Here again the above mentioned neuralgia was present on the first day of the attack and then gradually disappeared. All the symptoms of the previous attack repeated themselves, but they became more pronounced. The hallucinations tormented her considerably, so that the patient made repeated attempts at suicide. Equally the somatic symptoms were of a more marked character. The management of this attack was also more difficult than that of the first attack. The duration was also longer. It took six months for her to recover, but the termination was as abrupt as in the previous attack.

The family history shows that all her sisters are of a highly nervous temperament. An uncle died of paretic dementia and a grandmother died in an insane asylum. At the age of fourteen the patient presented for a short period symptoms of obsessions and agoraphobia, and at fifteen chorea twice during the same year.

Case 3. Mrs. R. P., aged thirty-four, had two attacks of melancholia, before she came under my care. Her husband, a man of intelligence, gives a clear account of her previous illness. After a confinement she developed a phlebitis, from which she recovered at the end of six weeks. During convalescence she suddenly became depressed. The condition grew worse and worse, so that she finally presented distinct symptoms of simple melancholia without delusions. The prominent symptoms were a pronounced depression, and indifference to the surroundings; the patient would spend her time in groaning and moaning, and lost interest in everything. The first attack lasted six weeks, after which the recovery came on abruptly and unexpectedly.

Six months later a similar attack occurred without any preliminary illness. The character of it was exactly like that of the first, but it lasted three months. Again the patient recovered suddenly.

I have had her under my observation for the last three years, during which time she had two attacks. The first of the latter also came on in the midst of apparently perfect health. Besides the painful emotional state, she presented also delusions of personal worthlessness and self condemnation, which at times led her to attempts at suicide. The condition, which lasted six months, was decidedly of a more serious nature than during the first two attacks.

After a recovery, which set in also unexpectedly, the patient remained in good health for eight months. After this interval of well being, another attack occurred.

This time she developed within a very short period a typical melancholia, with delusions of unpardonable sin; her sufferings, she says, are all due to her being untrue to the Lord. Her retribution, she says, is just and she must die for it. She is also tortured by frightful dreams, in which she sees her own body cut and burned by some invisible agents in punishment for her lack of religious ardor. There is no use, she says, to atone, as her wrongdoings were too numerous. Occasionally she has auditory hallucinations. She refuses food; spends her time in crying; lost all interest in her surroundings.

It is a little over a year that this attack has lasted, and the patient shows no sign of improvement.

The patient's family history is interesting. Three sisters were committed twice, an uncle on her father's side is a paranoiac. At the age of ten she had epileptiform convulsions.

Such is the brief account of three typical cases of intermittent melancholia.

The evolution of the disease, as well as other features, are so different from the common type of melancholia that they deserve a special mention.

One could see that in all of them there were no morbid symptoms immediately preceding the onset of each attack. While the ordinary melancholia develops in an insidious and progressive manner, so that the patient or his relatives cannot as a rule indicate the time when exactly the state of depression begins, in the periodic melancholia of the intermittent type the onset is strikingly sudden. Feeling perfectly well a day before, the patients awake in the morning with symptoms of sadness. Sometimes a localized physical pain will be present at the beginning, as in Case 2, in which each attack commenced with a facial neuralgia of short duration.

The duration of the attacks as well as of the intervals between the attacks presents no characteristic features, although some of the cases reported by other writers show a remarkable uniformity.

Inasmuch as the onset of the attacks is characteristic by its suddenness, their termination is equally abrupt or rapid, while an attack of ordinary melancholia disappears only gradually and very slowly.

As a last interesting observation, I wish to call attention to the state of mind of the patients between the individual attacks. It is singular to note the comparative mental lucidity, which is rather an unexpected phenomenon in view of the repetition of the attacks.

The cases which served as basis for the present study did not induce me to consider periodic insanities as a separate disease or to create a new form of melancholia. My intention was merely to present a modest contribution on an important variety of melancholia which has not received sufficient attention. I say *an important variety,* as it is not an indifferent matter for the patient and his friends to know whether he is suffering from an ordinary melancholia, which is, generally speaking, a curable mental affection, or from a variety of the same affection which has a tendency to repeat itself and become more and more aggravated with each new attack, and which by virtue of the repetition shows that there is a profound underlying degenerative make-up.

Krafft-Ebing,[2] from an extensive statistical study, shows how great is the rôle of heredity in periodic insanities. Although melancholia belongs to the group of mental affections in which a neuropathic tendency is frequently present, in the periodic variety of melancholia a mentally morbid heredity is still stronger. This fact creates a special state of degeneration, which predisposes strongly to periodic outbreaks of insanity. Ziehen's[8] careful statistical studies are in accord with Krafft-Ebing's.

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[2] Allg. Zeitsch. f. Psych. Bd. 26, 1869.

The three cases which I have just reported also show the degenerative tendency.

If this is the case, the prognosis in periodic insanities is certainly extremely unfavorable; the patient may recover from an individual attack, but the tendency to a repetition will be always present. This tendency is created by a special make-up of the mentality, which cannot be radically altered. Moreover, a careful reading of the records of others and my own cases showed to me that almost each subsequent attack presented a more serious clinical picture and was of longer duration than the one preceding. It is therefore evident that periodic melancholia is of a far more serious nature than the ordinary type of melancholia.

These few thoughts suggested themselves to me as being of some importance from a practical standpoint.

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[8] "Psychiatrie," 1894.

# Society Proceedings.

## NEW YORK NEUROLOGICAL SOCIETY.

April 7, 1903.

The President, Dr. Pearce Bailey, in the chair.

*Birth Palsy—Syringomyelia.*—Dr. Edward D. Fisher presented a man, twenty-five years of age, who had had a birth palsy, apparently the result of a tedious labor and a forceps delivery. There were present the characteristic athetoid movements, but the epileptic attacks had ceased in childhood. When fifteen years old he began to notice a wasting of the muscles of the left hand and forearm along with some disturbances of sensation. Subsequently the right hand became similarly affected. On examination, there was found to be almost complete loss of pain and temperature sense, and the tactile sense was not good. There was no sensation on either side to the strongest faradic current. There was absolute loss to both the faradic and galvanic currents in both hands. In the forearm there was a diminished reaction. The condition just described had progressed slowly during the past ten years. The reflexes were greatly exaggerated on both sides. The case had been previously diagnosticated as progressive muscular atrophy, but Dr. Fisher would make a diagnosis of syringomyelia, probably originating in a slight spinal hemorrhage. The patient was very intelligent and in perfect health. He had presented no bulbar symptoms.

Dr. B. Sachs thought the association of syringomyelia with an infantile cerebral palsy was unique. The suggestion of a hemorrhage occurring simultaneously into the cervical portion of the cord at the time of the cerebral palsy appeared to him a good one. Recently cases of congenital spastic paraplegia had been reported which were known to have been produced by hemorrhage into the cord lower down. The speaker said that a few days ago he had seen a case of apoplexy in an elderly person resulting in a left-sided hemiplegia. After a few days very slight motion returned in the left hand, and he was astonished to find that he could secure fairly good motion of the paralyzed hand when asked to close the other hand. This observation was common in children, but he had not before noticed it in adults.

Dr. Pearce Bailey said that a few years ago Schultze described hemorrhages into the spinal cord occurring in young infants in connection with difficult labor, and the subject had assumed a medico-legal interest. In the cases of hematomyelia that he had seen he had never been able to observe them long enough to see the progression of the symptoms.

*Suture of Popliteal Nerve; Perfect Restoration of Function.*—Dr. Russel A. Hibbs presented a boy of fifteen years who had come to the Orthopedic Hospital on October 6, 1902, with a history of having fallen upon a knife seven weeks previously and injured the leg. On October 9 he was operated upon, and on exposing the site of the injury it was found that the popliteal nerve had been divided. It was accordingly sutured with a practically perfect result. While the voluntary power in the muscles supplied by this nerve was lost, there was still some sensation, which was apparently accounted for by the fact that a few fibers of the nerve remained intact. The limb was kept slightly flexed for six weeks after the operation.

Dr. M. Allen Starr thought surgeons as a rule did not recognize the great importance of suturing nerves at the time of such injuries, for, it was not uncommon for persons so afflicted to seek relief a considerable time after the injury. There could be no question that a divided nerve would not unite spontaneously in the majority of cases, and it was equally certain that by nerve suture a good result was usually secured. He had seen one or two interesting cases in which nerve suture had been essayed in children in whom the brachial plexus had been injured at birth. Four successful cases of this kind had been recently reported. The operation consisted in laying bare the brachial plexus, finding what nerves were torn, and uniting them.

Dr. William M. Leszynsky said that in injuries of the hand special care should be taken to determine whether or not the nerves had been injured. He had recently seen a case in which the divided tendon had been sutured and the divided ulnar nerve had been entirely overlooked. As a result, a neuritis of this nerve had developed before he saw the case. Although rather late, an operation had been advised in the belief that even late operations were often successful.

Dr. Fisher said that he had had under observation for some time a facial paralysis that dated from childhood, the patient being a woman of thirty. After a while a faradic response from the facial muscles was obtained; hence he thought one could hardly place a time limit after which nerve suture would afford but little prospect of benefit.

Dr. Hibbs said that in his case the original wound had been treated at a hospital, but the nerve injury had not been noticed.

*Paralysis Agitans* (?)—Dr. J. Ramsay Hunt presented a man of forty-three, a cigar roller by occupation. The present affection dated back four years. It had first been noticed that the right hand was weak and tremulous, but he had continued at work for three years. Last July the head also became tremulous, and the right leg felt heavy. When first seen, about one month ago, examination showed tremor of the right hand without rigidity; a slight atrophy of the right forearm; a tendency to monotony of speech and a rather expressionless face. The question arose as to whether this was an occupation-atrophy or, as he was inclined to believe, a case of paralysis agitans.

Dr. Hunt also reported a case at the Montefiore Hospital which had presented the characteristic picture of paralysis agitans for several years before death. An autopsy on the brain was alone permitted. The vessels showed marked arteriosclerosis. There was an excessive degree of pigmentation and some rounding of the processes of the cells in the cortex and in the portion of the cervical cord removed for examination—in short, those changes that had been regarded as occurring frequently in paralysis agitans. The specimens were exhibited under the microscope.

*Elephantiasis Ossium.*—Dr. I. Abrahamson presented a woman of thirty-six showing a peculiar glossy appearance of the skin of the forehead and a remarkably wide supraorbital ridge. Examination revealed a ptosis of the right eye, a double divergent squint, and a marked contraction of both inner fields of vision. According to her husband, the changes in her outward appearance had been progressive. The malar bones were enlarged, and apparently the hands and feet were growing larger. The condition was possibly due to luetic disease of the apophysis, because there was specific disease of the eyes.

Dr. Starr remarked that the appearance of the eyebrows pointed strongly to the case not being one of acromegaly or myxedema, but of elephantiasis ossium. The case appeared to be in an early stage.

Dr. Leszynsky said that acromegaly could be excluded by the condition of the visual field, as in that disease there was usually a bitemporal hemianopsia.

*Paralysis Agitans. A General Discussion.*—Dr. B. Sachs said that he was of the opinion that this was distinctly a disease of the senile period. In this period the two most important factors were emotional excitement and some prolonged and exhausting disease. The point of differential diagnosis to which Charcot had attached so much importance, the non-involvement of the head in paralysis agitans and its involvement in multiple sclerosis, seemed to him a point not well taken, for he was sure he had seen involvement of the head in fully 75 per cent of cases of paralysis agitans. Charcot's attempt to differentiate sharply between these two affections seemed to him to have been carried beyond a justifiable limit. He had seen cases giving the symptoms of multiple sclerosis, and yet later on develop a distinct picture of paralysis agitans. There was a young man at the Montefiore Hospital whom he had first seen when only about eighteen years old, and who developed the typical carriage, tremor and speech of paralysis agitans. As the years went on he developed nystagmus and a marked exaggeration of all the reflexes, and his speech was now as much that of multiple sclerosis as of paralysis agitans. He also recalled the case of a young woman first seen by Professor Mendel in Berlin, who made the diagnosis of hysterical tremor. When seen by the speaker a few months later, she presented what appeared to be the first symptoms of multiple sclerosis. At the present time, she presented the typical picture of paralysis agitans. This experience led him to think that there must be some relationship between these two diseases, and the case presented this evening by Dr. Hunt gave color to this view. With regard to the treatment of paralysis agitans he was accustomed to make use of hyoscine and the ordinary sedatives, and probably all present believed that no drug did much good. The only treatment that was fairly satisfactory was the use of some form of vibratory therapeutics.

Dr. Stuart Hart said with regard to the records of Dr. Starr's clinic at the Vanderbilt Clinic, that out of 219 cases of paralysis agitans there recorded, 139 were males and 80 females. Under thirty years of age there developed 2 cases, both males. The disease commenced in 88 cases between fifty and sixty years of age. Among heavy workers there were 45 males; among those particularly exposed to cold there were 24 cases; among the lighter trades there were 24 cases; among those working in factories were 14 cases. In 6 cases it was stated that the mother had paralysis agitans, and in 3 the father was said to be so affected, while in several other cases brothers or sisters were said to be similarly affected. Forty patients believed that the disease arose from anxiety or worry, and half of these directly attributed it to fright. In quite a number of instances the history stated that the tremor had developed very shortly after some trauma of the part first exhibiting the tremor.

Dr. Joseph Collins presented an analysis of 50 cases of paralysis agitans. Of this number 34, or 68 per cent, were males, and 16, or 32 per cent, females. The average age at which the disease developed was 51½ years, the youngest being 32 and the oldest 72 years old. They were divided according to occupation as follows: Merchants, 7; workmen, 8; laborers, 5; tailors, 4; clerks, 1; clergymen, 1; drivers, 1; carpenters, 1; plasterers, 1; engineers, 2; gardeners, 1; houseworkers, 9; captains, 1; no occupation, 7. The nationalities were: United States, 8; England, 1; Ireland, 16; Germany, 7; Holland, 1; Russia, 6; Austria, 1; not given, 10. The attributed cause was: Worry in 7 cases; alcohol, 1; excesses, 1; excitement, 1; shock, 1; refrigeration, 1; hard work, 1; pneumonia, 1; grief, 1; bite of cat, 1; unknown, 34. In 7 cases there was a history of injury; in 1 of operation; in 1 of hard work; in 1 of syphilis; while in 40 cases there was no special history of this kind. There was a history of neuropathic heredity in 13 of the cases, or 26 per cent, and a history of paralysis agitans in the parents or uncle in 6 instances. The average duration when first seen was four years, the longest being 20 years, and the shortest 5 months. The part to first show trembling was, left upper extremity in 11 cases; left lower extremity in 8; right upper extremity in 15; right lower extremity in 1; upper in 7; lower in 2; right upper and lower in 1; left upper and lower in 1; and all extremities in 4 cases. The initial symptom was: trembling in 24 cases; pain in 11; unsteadiness in 3; numbness in 2; nervousness in 1; stiffness in 1; inactivity of fingers in 1; weakness in 1; loss of power of right hand in 1; headache in 1; and not given in 4 cases. The relation of curvature of the spine was investigated specifically in only the last 20 cases, and was noted in 7 cases. He was convinced that paralysis agitans was a disease of early senility occurring as the reward of virtue.

Dr. M. Allen Starr said that he had had a fairly large experience with paralysis agitans, yet it did not bear out Dr. Collins' statement about the frequency of the disease among the Celts, and its comparative rarity among Hebrews. He could recall quite a large number of patients among Jews, and a number among the Germans. In hospital and clinical work one was apt to get a one-sided view of such matters, because of the large proportion of persons of certain nationalities at such places. Nor did his observations bear out what the last speaker had said about heredity; probably four or five per cent would represent the proportion showing hereditary influence. Anxiety and trauma appeared to him to bear a distinct relationship. He had never seen a patient absolutely cured of paralysis agitans, but he had observed a very marked improvement in one, and a great variability of symptoms in a number of other cases. The disease did not appear to be a steadily progressive one. He had found that the Swedish massage, skilfully administered, after a prolonged hot bath, gave these patients so much relief that they were willing to keep up such treatment for years. The great majority of his patients had found benefit from hyoscine in doses of 1-100 of a grain, given from two to five times a day.

Dr. Leszynsky said that dispensary and hospital patients were not benefited, but with private patients it was quite different. Four times within the past ten years he had had the opportunity of making the diagnosis very early in the disease. The disease certainly presented remissions at times, and traumatism certainly exerted a distinct influence. Most of these patients were very amenable to suggestion, and in this way could be benefited by treatment. He had not observed good effects from hyoscine except for a very short time, because most of the patients complained of the action of this drug. As systematic exercise had been recommended he had tried the Swoboda system of exercises, which call for voluntary effort entirely, and the patients appeared to be benefited thereby.

Dr. Joseph Fraenkel said that in the past ten years he had seen many cases of paralysis agitans and had witnessed 10 autopsies. It was rare to find a person suffering from paralysis agitans who was gray-haired or old-looking, and the autopsies showed a remarkable freedom from arteriosclerosis. These persons were rarely addicted to the use of alcohol or exhibited evidence of syphilitic infection, and to this extent he would indorse Dr. Collins' statement that this disease was "the reward of virtue." He saw nothing to support the theory that this was a disease of senility. It was common to oberve a marked hypertrophy of the skin in these patients. A characteristic feature was the difficulty of stopping the patient when on any one line of thought and changing him to another. A pseudonystagmus was often observed. Although the tremor ceased at night, the paralysis agitans patient presented the same attitude in sleep as when awake. He agreed with Dr. Collins that very little could be done for these patients by treatment of any kind.

Dr. Harlow Brooks said that he had only studied four cases of paralysis agitans post-mortem, and the findings were so conflicting that he was very skeptical about any lesions claimed to be characteristic of the disease. He was of the opinion that deformity of the spinal column was very common in persons who were not very well developed, basing this opinion upon many observations made in the deadhouse as well as in examining during 1898 the recruits for service in the regular army. He thought that fully 7 out of 50 such recruits would show as much spinal deformity as Dr. Collins' cases, even though these persons were of sufficiently good physique to be accepted in the army.

Dr. Collins said that his statistics with regard to the Celts might be refuted, but to do so the actual figures must be presented and not cases recalled from memory. He believed also that the more paralysis agitans was treated the worse it became.

Dr. Hunt said he understood that the premature senility referred to the nervous system, and not to the arterial tree when used in connection with paralysis agitans.

Dr. Pearce Bailey was of the opinion that two-thirds of the cases of paralysis agitans began after the age of sixty. A disease which could become fairly well developed in the course of a few weeks could hardly be regarded as an organic disease of the nervous system, and he was inclined to think that the evidence was rather in favor of its being a disease of the muscular system, resulting possibly from the action of certain toxins. He believed it was distinctly induced by trauma, either psychic or physical, for there were too many definite and striking clinical records of this kind. The disease usually first appeared in the hands, and yet in cases of injury to the legs the disease was more apt to begin in the legs. A large proportion of the persons coming to Dr. Starr's clinic suffering from paralysis agitans were apparently right-handed workers.

*A Specimen of Brain Abscess.*—Dr. Joseph Collins presented this specimen removed from an Italian laborer, forty years old, who had not been sick up to two months before his admission to the City Hospital, since which time he had suffered from headache only. On January 14 he became stuporous. There was no paralysis, paresis or spasm, in fact, no objective symptom other than stupor. Examination on February 3 showed the head to be drawn over to the right side and flexed, and the right upper extremity partly flexed at both joints. There was a peculiar elastic resistance of the right upper extremity on extending it. Knee-jerks were absent. The plantar jerk on the right side was lively, but could not be elicited on the left side. The body temperature remained about normal, and the pulse varied from 68 to 90, and the respirations between 20 and 22. Dr. Collins' diagnosis was circumscribed non-purulent encephalitis, and because of the motor symptoms on the right side the lesion was thought to be in the left cerebral hemisphere. However, the lesion was found to be a focus of comparatively recent yellow softening situated on the right convexity, involving that portion of the first temporal convolution lying immediately below the supramarginal and angular gyri, and separated from these by the Sylvian fissure. It involved the posterior arm of the internal capsule, and extended apparently into the extraventricular radiations emanating from the lower third of the posterior central gyrus. The internal capsule was not involved in its passage between the basal ganglia. No thrombus could be found. The special interest of the case lay in the fact that the motor symptoms were on the right side and the lesion was found on the same side.

# Periscope.

### MISCELLANY

*Cardiac Neuroses.*—Pick, in his capacity as the chief of the Vienna Military Hospital, observed 41 soldiers between eighteen and thirty years of age, with a syndrome of symptoms approaching the character of Basedow's disease, but at the same time differing so essentially from it as to merit special consideration. The patients suffer from palpitation and oppression at the least effort, and complain of muscular fatigue. At the physical examination they all presented symptoms of a certain degree of a hypertrophy of the thyroid gland, usually of the left lobe, but the goiter was never pulsatile. The pupils are unequal, the right one rather dilated, but reacting to light; the mydriasis can hardly be ascribed to pressure of the right sympathetic through the lobe of the thyroid for the hypertrophy of the latter is too insignificant to cause such disturbance. All the subjects suffer from a very irritable heart, and the slightest effort suffices to increase abnormally the heart beats; so that in one minute the beats will jump from 70 to 140 per minute. The heart, as asserted by Kraus in other cases of thyroid hypertrophy, is rather dilated than hypertrophied. The cutaneous capillaries are very irritable and all the patients present marked dermography. Axillary hyperidrosis, anesthetic pharynx, but without other symptoms of hysteria. Some show tremor of hands when extended with eyes closed, others some cardiac arrythmia. Unable to form a positive diagnosis the author supposes the syndrome of symptoms to be a disguised form of Basedow's. JELLIFFE.

*The Various Reflexes in Diabetes.*—It has been observed by Landouzy Rosenstein and others that the patellar reflex is either diminished or entirely absent in cases of diabetes mellitus. Pitres (*Soc. de Bul. de Bordeaux,* Nov. 11, 1902) undertook to investigate the condition of other reflexes in this affection. With this in view he examined 32 diabetics, and he finds the following:

| | *Patellar.* | *Cutaneous.* | *Cremaster.* | *Plantar.* | *Pupillary.* |
|---|---|---|---|---|---|
| Abolished .......... | 13 | 6 | 19 | 16 | 1 |
| Weakened .......... | 7 | 8 | 6 | 2 | 0 |
| Exaggerated ........ | 2 | 6 | 4 | 6 | 0 |
| Normal ............ | 10 | 1 | 3 | 8 | 31 |

It would thus seem that the cutaneous reflexes are, even more often than the patellar, either abolished or weakened. At times the tendon reflexes are first to disappear, at other times it is the cutaneous; of the latter we find at one time the cremasteric weakened or abolished, at other times the abdominal or the plantar, while the others are either exaggerated or normal. This great variability is observed also in other pathological states, especially in tabes, and this renders it somewhat difficult to explain the significance of the loss of reflexes. Contrary to the above, the pupillary reflexes are almost always preserved in diabetes. In the single case in which the author found them absent there was arteriosclerosis, diabetes with glycosuria and moderate polyuria. In the other cases the reaction of the pupils to light, accommodation and pain were perfectly normal. The conclusion may thus be drawn that although the condition of the cutaneous and tendon reflexes may not seem in the way of a differential diagnosis between true tabes and diabetic pseudotabes, the character of the pupillary reflex and of the epigastric sensibility as well as the cremasteric reflex may be utilized in difficult cases to distinguish one affection from the other; inasmuch as the Argyll-Robertson pupil and the epigastric and testicular analgesia, so common in true tabes, are not observed in diabetes.

JELLIFFE.

### RIVISTA SPERIMENTALE DI FREMATRIA

(Vol. xxviii, Fasc. iv, Dec., 1902.)

1. The Correlation between Psychic and Organic States. SILVIO TONNINI.
2. A New Anomalous Process in the Sphenoid Bone of Man. TENCHINI and ZIMMERL.
3. Peripheral Facial Diplegia. PANEGROSSI.
4. A Contribution to the Study of Precocious Malignant Syphilis of the Brain. PANICHI.
5. The Toxic Principles of *Aspergillus Fumigatus* and *Flavescus* and Their Relation to Pellagra. CENI and BESTA.
6. The Nervous Lesions and the Pathogenesis of Amyotrophies of Articular Origin. PIGHNI.
7. A Contribution to the Study of Lingual Hemiatrophy. BIANCONE.
8. The Toxicity of the Blood of the Insane. CENI and PINI.
9. Hemorrhagic Encephalitis with Special Reference to the Tuberculous Variety. BOMBICCI.
10. Hypothermy in the Epileptic. BESTA.
11. The Assistance Rendered to Insane in Italy. TAMBURCNI.
12. The Action of Gastro-intestinal Juice on *Aspergillus*. CENI.

1. *The Correlation between Psychic and Organic States.*—The author employs the term correlation to express that close and varied connection which unites and co-ordinates the organic functions with the psychic ones, and these last with the psychic acts as manifested to the eye, etc. Disease, degeneration, mental sanity have all their proper psychic and psycho-organic correlations, and these correlaries are to be found not solely in pathologic conditions; for correlation is a biological function, and without it there is no life. To give an idea of the importance practically of these organic and psychic correlations, it is sufficient to take some examples from the sphere of the sexual life which exerts such a direct influence on the development and orientation of psychic life. Thus sexual perversions are always associated with certain organic and psychic secondary sexual characteristics. The author brings forth a case in point, that of a man of fifty-seven who was accused of seducing a young woman, but who claimed to be impotent. Physical as well as a thorough psychological examination by the author concerning all the man's habits, mode of thinking, etc., proved to the jury conclusively that the man could not but have been perfectly potent. The author makes the proposition that psychological examination and investigation should be carried out not only in cases of suspected mental alienation, but in all cases that lack proof of a committed crime; as such an examination requires knowledge and skill it should generally be entrusted only to an experienced psychiatrist. This province has, however, been invaded to some extent by Criminal Anthropology, with its practical tendencies to fix the correlation between the physical and psychical character of certain criminal types. Thus the mobile physiognomy, the erratic look, the uncertain gait of the thief are but dynamic symbols approaching the author's psycho-organic correlations. A great importance is to be attached to gesture, to various signs and motor phenomena which very frequently serve as substitutes for language. The author brings forth examples

from the creations of the great writers of the present time (as Tolstoi. Victor Hugo, Zola, Bourget, etc.), showing how their deep psychological insight into human nature enabled them to comprehend and present in their works the correlation between the physical and psychic states of the presented types.

2. *A New Anomalous Process of the Sphenoid Bone in Man.*—Of interest to the student of anatomy.

3. *Peripheral Facial Diplegia.*—Double paralysis of the facial nerve of peripheral origin, although known to Christison (1835), Bell (1836), and others, is of rather rare occurrence, and quite difficult to diagnosticate; nor is the etiology of the affection always easy to determine. The author describes three cases with an undoubted clinical picture of the affection. What strikes the observer in all the three cases is the fixed look and the peculiar aspect of the immobile, expressionless, mask-like face, the perfectly smooth forehead. the eyes open wider than normal, the nostrils fallen in, the naso-labial sulcus smooth, the cheeks flattened, the lower lip hanging down. The patients were unable either to frown, or to close their eyes; could neither spit. whistle nor blow, and also found it difficult to pronounce certain letters—all this showing that the motility of all the muscles of the face that serve mimicry was completely abolished. In differentiating this paralysis from a central nuclear affection of the nerve the author considers first the fact that both the inferior as well as the superior facial were involved in the paralysis; secondly in one of the cases the paralysis was localized exclusively in the area occupied by the seventh nerve, an occurrence which is frequent in paralysis of peripheral origin. but rather rare in that of nuclear affection, in which together with the facial there are also attacked other cerebral or spinal nerves. As distinguished from a labio-glosso-pharyngeal paralysis it is to be noted that this last, with very few exceptions, does not extend to the upper part of the face, nor is mimicry in any way affected, and while in peripheral facial diplegia both sides of the face may be attacked simultaneously, in the labio-glosso-pharyngeal paralysis the lips suffer first, and then progressively the tongue, the palate, the larynx, etc., muscular atrophy ensues alongside of the paralysis and both advance slowly. The slowness in the advance of the disease, and the early atrophy as found in superior polioencephalitis serve to exclude this malady; moreover. in this affection the paralysis of the facial muscles is always associated with that of the muscles of the eye, a rather rare occurrence in facial diplegia. Atrophic paralysis of the muscles of the face is also observed in certain cases of progressive muscular atrophy of the juvenile type of Landouzy-Déjerine. But the whole facial aspect of these cases is different from that presented by the patients in question, and this facial appearance is but an insignificant part of the clinical picture of the affection; moreover, in these cases the atrophy and paralysis of the muscles of the face is even of a slower development than in labio-glosso-laryngeal paralysis. From the time of Christison (1835) up to Davanil (1850) there was but one monograph in the literature of this affection; next we find Wachsmuth (1864), who was the first to understand the disease better than his predecessors and who gave to it the name of diplegia. He was followed by Pierreson in 1867, and then of late Stintzig (1893), Hirbschman, Bregman, and quite recently Raymond, have all devoted considerable time to this disease. The author presents an elaborate and very instructive summary of ninety cases of this affection, so far found in literature, with the designation of the symptoms, course, etiology and termination of each. This summary is valuable enough to present by itself a full monograph on the subject.

4. *Malignant Syphilis of the Brain.*—The patient, a man of twenty-

seven years, was admitted to the hospital with paresis and a beginning disturbance in the motility of the tongue. This condition seems to have ensued rapidly during one night; and to it were soon added paralysis of the left half of the face, conjugate deviation of the eyes to the right; continuous yawning. The anamnesis of the patient is somewhat indefinite, except that he became recently a hard drinker, and contracted syphilis over a year previous to admission; two months previous to this admission he began to suffer with irregular fever, and vague pains in the articulations; more recently with intense headache, of a diffuse and continuous character. During observation in the hospital the cardiac symptoms (formerly mitral insufficiency) grew worse, and the frequency of the heart beats increased up to 130 and 160 per minute. Notwithstanding the energetic anti-syphilitic treatment resorted to while in the hospital the improvement was very insignificant. A month before the fatal issue the condition of the patient was as follows: Patient prefers to lie on right side with head and eyes turned toward same side; when grinding his teeth the left corner of mouth remains immovable; in frowning the left side of forehead is almost flat; eyes present some nystagmus. The tongue is deviated toward the left, but is quite mobile; shows some slight fibrillation. The left upper extremity is flexed at a direct angle and lies near the trunk, but preserves voluntary as well as passive motions. The left lower extremity is bent at an obtuse angle to the knee, admits of no movement whether active or passive. Patient urinates freely, but the function of defecation is somewhat affected. Left pupil wider and less active than the right. All the reflexes abolished on the left side; thermic and tactile sensibility considerably diminished on same side; hearing also abolished on same. Bilateral hemianopsia of the right halves of both retinæ. The patient remained almost all the time in a state of sopor, from which he would awake with some difficulty. The anatomical diagnosis on autopsy was as follows: A dissecting aneurism of the second branch of the right Sylvian artery (middle cerebral); aneurism of the fourth branch of the left Sylvian; softening of the gyri of dextra insula, and hemorrhage in the right capsule of the thalamus. The microscopic examination of the lesions established beyond doubt the syphilitic origin of the disease (syphilitic arteritis, syphilitic aneurism).

5. *The Toxic Principle of Aspergillus.*—An experimental study on the toxicity of *Aspergillus fumigatus.*

6. *Amyotrophies of Articular Origin.*—A continued article.

7. *Hemiatrophy of the Tongue.*—A continued article.

8. *The Toxicity of the Blood of the Insane.*—A rather unsatisfactory account of a series of experimental studies to determine the toxicity of the blood of patients suffering from various forms of insanity. The authors are forced to confess that notwithstanding the considerable number of experiments it was impossible to establish definitely any difference between the serum of normal individuals and that of insane. Whatever toxicity was demonstrated was observed in the blood of maniacs and epileptics. The toxic power of the blood of individuals affected by various psychopathic maladies (maniacs, alcoholics, paralytics, paranoiacs, epileptics, etc., did not present any notable variations; nor was there any variation in the different forms of one and the same disease, as in the periods of calm and excitement. It would seem as if the present methods of experimentation were not sufficiently reliable to enable one to control and definitely establish the degree of toxicity of the blood serum in the various maladies in question.

9. *Hemorrhagic Encephalitis.*—A continued article.

10. *Hypothermy in Epileptics.*—The case serving as the basis or this

observation was that of an epileptic, twenty-eight years of age, who had been suffering from epilepsy for the last ten years. During the last two years the attacks occurred four or five times a month and were followed by a state of depression and mental confusion. Patient is rather of a taciturn disposition, but well physically and without any degenerative stigmata. Nutrition good, functions of organic life normal. The morning temperature was 35.2 deg. and kept low for about 18 days; during this time there occurred oscillations between 35.1 deg. and 36.4 deg. There was no symptom to indicate any abnormal condition or even slight disturbance whether physical or mental. The author ascribes this low temperature to some special condition of the metabolic processes of the epileptic. It is quite possible that the same toxic agent which acts on the nervous system producing the condition of epilepsy may be the cause of the hypothermy in these cases.

11. *The Condition of the Insane in Italy.*—An historial essay on the present condition of the asylums for the insane in Italy, showing a recent increase in the number of the insane population of the land, and an overcrowding of the institutions for the maintenance of the insane. The author proposes various provisions for relieving the congestion in the asylums, and especially a system of family care of the insane.

12. *The Action of the Gastro-Intestinal Juice on the Spores of Aspergillus.*—This is a complement to article 5, on the relation of the spores to the genesis of pellagra. ALEX. ROVINSKY (New York).

## NEUROLOGISCHES CENTRALBLATT

(1903, No. 3, Feb. 1.)

1. The Isthmostriatic Tract (or Bulbo-striatic Tract) of Pigeons. A. WALLENBERG.
2. A Remarkable Complication of a Case of Migraine. L. HOEFLMAYR.
3. A Contribution to the Knowledge of Disseminated Encephalomyelitis. E. BANCKE.
4. Ear Reflex. W. ALTER.

1. *Isthmostriatic Tract.*—Wallenberg failed to obtain degeneration of the isthmostriatic tract in a pigeon by unilateral disturbance of the cerebellum even if the central nuclei of the cerebellum were involved. Division of the sensory root of the fifth nerve between the bulb and the Gasserian ganglion resulted in degeneration of the dorsal and lateral periphery of the nuclei. It was then possible by the Marchi method, to trace the tract as far as the anterior portion of the base of the brain. In the frontal lobe this tract of fibers divides into four bundles, which terminate in different parts. The terminal sensory trigeminal nucleus, therefore, is in direct union with the basis of the frontal lobe without the intermediary of the thalamus.

2. *Migraine.*—A woman, fifty-seven years of age, who had ceased to menstruate six months before. Father and sister had suffered from migraine; one of her children was hysterical and had headache. Patient had migraine for forty years. Lately attacks were more frequent and constipation and the menstrual flow would aggravate symptoms. Had taken large doses of migrarine every day previous to special attack. This attack began with a pulling up of one eye and one eyeball, hemiatropia, collapse and the fear of death. She was semiconscious for several days without sleeping. When disturbed would repeat the syllables "noten" and "nieten." There was obstinate constipation and agrypnia. On the sixth day the face assumed an expression of pain. Twitchings of both arms and legs appeared. During sleep she was quiet. On the tenth day consciousness returned, after

which she improved slowly, but had occasional hallucinations and some disturbance of the field of vision. The symptoms were those of ophthalmic migraine and toxine infection. It is possible that coprostasis was the exciting cause.

3. *Disseminated encephalomyelitis.*—To be continued.

4. *Ear Reflex.*—Alter describes a new skin reflex which is obtained by striking the ascending ramus of the inferior maxilla resulting in a vigorous contraction of the attoleus aurem with elevation of the ear. He found this in a paretic dement with left-sided facial paralysis and status epilepticus, by striking on the right side. On investigation he found it 19.4 per cent of patients with paretic dementia. It differs considerably on the two sides. Alter does not arrive at a definite conclusion regarding its meaning.

(1903, No. 4, February 15.)

1. A Contribution to the Casuistry of the Relation of Hair in Cases of Mental Diseases. W. Heinicke.
2. The Biology and the Functional Activity of the Central Nerve Cells. P. Kronthal.
3. A Contribution to the Knowledge of Disseminated Encephalomyelitis. E. Bancke.

1. *Hair in Mental Disease.*—Henicke reports the case of a girl twenty-one years of age suffering from dementia præcox. Symptoms had first appeared at the age of thirteen. She had occasional periods of excitement, in one of which it was observed that the pupils were dilated ad maximum and that an almost silver white band of hair passed from the forehead to the right parietal bone. The hair remained in this condition for four days and then rapidly returned to its normal condition. Three days later a second attack occurred, accompanied by a similar dilatation of the pupils, but the coloration of the same band of hair was observed one to two hours before attack. During the height of the attack the hair became almost silver white. The following day it returned slowly to its normal blonde color. Microscopical examination showed that this change of color was due to the filling up of the sheath with air, and when the air was expelled, the hair returned to its normal color. The manner in which the air accumulates cannot be determined.

2. *The Central Nerve Cells.*—Kronthal begins with a consideration of phenomena which follow stimulation of the gray matter of the cortex. These he considers as due to the excitation of the nerve fibers. It is noteworthy that changes of diet do not affect the nerve cell, and that the central nervous system, even in starvation, suffers less than any other part of the body. Some substances, as narcotics, act upon nerve cells as upon other forms of protoplasm. Ganglion cells in the process of division have never been found. The hypothesis that man is born with a large mass of reserve cells he assumes as impossible. Kronthal occasionally has observed a strange cell in the nerve cell which he thinks is a leukocyte. Kronthal believes, therefore, that the nerve cell is due to a coalescence of leukocytes, that it is no organism, and that it is not alive. The functions of the nervous system are accomplished by the neurofibrils which unite the nerve fibers. The functions of the nerve cells have not been definitely determined.

3. *Disseminated Encephalomyelitis.*—A school teacher, thirty-nine years of age, with mental symptoms of dementia præcox, had meningitis in childhood. Five and one-half years before death she began to limp on left side. Examination showed excessive spasticity and contractures in left leg, the latter disappearing completely during chloroform narcosis. A plaster cast was put on, which, after 17 days, had to be removed on account of extensive decubitus. About a month later paralysis of the bladder and

rectum and lower extremities, with loss of sensation, appeared. Later still paralysis of the right arm. After four months she died. There was a fresh internal hemorrhagic pachymeningitis. Microscopically numerous areas of degeneration in various stages were found throughout the brain and cord, which are described in detail. A secondary gliosis was present which was characterized by the presence of large cells with oval nuclei. Bancke believes that the case was one of dementia præcox in which infection was produced by the bedsore. T. H. WEISENBURG (Philadelphia).

### MONATSSCHRIFT F. PSYCHIATRIE UND NEUROLOGIE

(1903, April.)

1. A Case of Isolated Agraphia. C. WERNICKE.
2. Hyperesthesia of the Nails (Onychalgia nervosa). H. OPPENHEIM.
3. Epileptic Mania with Remarks upon the Loss of Ideas. H. HEILBRONNER.
4. The Origin and Course of the Oculo-Motor Nerve in the Mid-Brain. N. MAJANO.

1. *Isolated Agraphia.*—A woman forty-six years of age noticed a gradually increasing weakness of the right hand which suddenly increased to paralysis of the right arm and leg with contractures. Eight days later there were twisting of the mouth, and complete loss of speech, although the patient appeared to understand what was said to her. Gradually speech improved and then grew gradually worse and remained stationary. At this time there was paralysis with spasticity of the arm and leg, anarthria, good repetition, and some spontaneous speech. She understood words fairly well, but there was complete agraphia. Memory was defective and the patient remained in this condition during all the period of observation. Therefore the predominant symptom and the only complete one is the isolated agraphia. Only a few similar cases are reported in the literature. The characteristic of all these cases is that the lesion is on the left side. Nevertheless, Wernicke does not share the opinion of Dejerine that there is a unilateral optic word center, at least he does not believe that the existence of such a center is proved by these cases.

2. *Hyperesthesia of the Nails.*—Oppenheim reports three cases characterized by extraordinary sensibility of the finger-nails so that even slight mechanical injuries caused intense pain. Even trimming the nails caused pain. In all cases there was congenital neurasthenia. There are a few references to this condition in the literature, although it does not appear to have been recognized as a particular form of hyperesthesia.

3. *Epileptic Mania.*—Heilbronner continues his article with a discussion of his cases. He calls attention to the undoubted false perception that sometimes occurs in epileptics, and then commences to discuss the flow of ideas. He does not believe that there is any necessary relation between these and the readiness of speech, in fact in many cases speech may be distinctly inhibited although the patients complain of a continual series of thoughts passing through their heads; that is to say, there may be a flow of ideas without any motor excitement. It does not appear that the flow of ideas is the result merely of an increased psychological motor irritation, not even in mania. In this condition there is motor excitement, but the flow of ideas is independent of it. It is always important to compare the inclination to speech with the psychical representations that the patient has prepared. It must be admitted, however, that the whole subject is in a very unsatisfactory state.

4. *Oculomotor Nerve.*—Majano continues his article with a careful study of the literature of the origin and course of the oculomotor nerve.

He concludes partially as a result of these studies, and chiefly from his own experience, that the sublongitudinal or predorsal fasciculus has its origin in the anterior nucleus of the anterior corpora quadrigemina; that the ventral portion of Meynert's ligament arises from the dorsal median region of the copora quadrigemina; that the sublongitudinal fasciculus passes obliquely forward and downward toward the median line, enters the capsule of the red nucleus, and some of the fibers then pass down to the root fibers of the oculomotor, but the main portion decussates and unites with the root fibers of the oculomotor of the opposite side. The mechanism of the pupillary reflex then would consist of the following three parts: the peripheral sensory neurone arising from the bipolar cells of the retina and forming the pupillary fibers of the optic nerve which pass to the dendrites of the ganglion cells of the lateral portion of the anterior corpora quadrigemina. Second, the central neurone which consists of the ganglion cells of the lateral portion of the anterior corpora quadrigemina, and the direct or crossed fibers of the predorsal fasciculus which unite with the root fibers of the oculomotor nerve, and with the collaterals of the axiscylinder and sympathetic cells of the ciliary ganglion of the same or the opposite side; third, the peripheral motor neurone, which consists of the cells of the ciliary ganglion and the fibers arising from them which innervate the sphincter of the iris. The pupillary reflex is, therefore, primarily dependent upon the anterior corpora quadrigemina of the opposite side. The direct pupillary reaction is stronger than the indirect on account of the double decussation of the neurones concerned in it. The unilateral reflex paralysis is more pronounced on one side, occurring when the neurones between the anterior corpora quadrigemina and the ciliary ganglion are degenerated. If the ciliary ganglion and the ciliary nerves are destroyed then total paralysis occurs. The consensual reaction is not destroyed by longitudinal section of the chiasm. J. SAILER (Philadelphia).

## DEUTSCHE ZEITSCHRIFT FUR NERVENHEILKUNDE

(Band 22, Heft 3-4.)

12. Skin Reflexes and Their Nerve Tracts. MUNCH-PETERSEN.
13. Contribution to the Knowledge of Tumors of the Spinal Cord. MEYER.
14. Multiple Sclerosis Developing into a Picture of Transverse Myelitis. FLATAU and KOELICHEN.
15. Acute Cerebral and Cerebrospinal Ataxia. LÜTHJE.
16. Contribution to the Diagnosis of Tumors in the Frontal Lobe. AUERBACH.
17. A Case of Relapsing Bilateral Ptosis, with Myasthenic Manifestations in the Upper Extremities. BYCHOWSKY.
18. Cystic Meningitis in Racemose Cysts of the Central Nervous System. ROSENBLATH.

12. *Skin Reflexes and Nerve Tracts.*—Munch-Petersen contributes a valuable article upon the reflexes of the skin, first discussing the history of some of the more important reflexes, and then defining them as involuntary movements which are produced by irritation of the skin, he then speaks of the atypical form which can be obtained in almost any part of the body, and then mentions the so-called typical forms which include the plantar, cremaster and abdominal reflexes. The degree of irritation required indicates inversely the activity of the reflex. There seems to be some relation between the skin reflexes and the tickling reflexes. He believes the plantar reflex has a single reflex center; discusses most thoroughly the Babinski phenomenon, calls attention to the fact that there is dorsal flexion of the toes during walking in persons who possess it; and discusses some

interesting time estimations that he made upon this reflex by the aid of kinometographic studies. Investigations have convinced him that the movements of the toes are irregular when the soles of the feet are irritated. He believes that there is a distinct relation between the gait and the plantar reflex, that is to say, they are typical or atypical together. With regard to the arcs by which these reflexes are accomplished, he makes the following statements. When sensation is lost the reflex is lost; when increased the reflex is increased; therefore, sensation is an essential condition for the reflexes. If, however, the reflex is produced by sensation, the sensitive part of the reflex arc must have its center in the cortex of the cerebrum. The relation of the plantar reflex to the gait indicates that it has some relation to a motor center in the Rolandic cortex. Analogy indicates that the skin reflexes have their sensitive as well as their motor centers in the cortex of the cerebrum. A study of these reflexes in different conditions seems to lead to the same result. In diseases of the peripheral nerves or in total lesion of the spinal cord the skin and tendon reflexes were entirely lost. Voluntary motion and sensation were also lost, and, therefore, there was reason to believe that the tracts of these different functions were identical as far as the point of solution of continuity. Munch-Petersen has found that when the plantar reflexes were in part preserved the motor tracts were not entirely destroyed. Studies of apoplexy also lead to the conclusion that at least as far as the internal capsule, the reflex tracts and the tracts for voluntary motion were identical. In cases of recent apoplexy with a persistent plantar reflex the prognosis is more favorable. Further, in those cases in which there is ataxia in motion, dorsal flexion during walking is found in those cases in which the Babinski phenomenon is present. Babinski's phenomenon he believes is due to a degeneration of the pyramidal tracts which gives rise to a paralysis of the plantar flexures or to ataxia. In hysteria the skin reflexes are influenced, but show no qualitative alterations from the normal. During hypnosis suggested anesthesia diminishes or abolishes the skin reflexes; suggested hyperalgesia increases them. In conclusion Munch-Petersen calls attention to the following experiment. If the sole of the foot is irritated with a needle the foot is drawn upward; if a threat is made to irritate the sole of the foot with a needle, the foot is also drawn up, finally in some cases the mere sight of the needle is sufficient to produce an involuntary movement of the foot. Therefore, we cannot state certainly that the skin reflexes are not produced by an act of the will, but only that this activity of the will does not reach the highest phase of consciousness. The skin reflexes are in all probability located in the cortex of the brain.

13. *Spinal Cord Tumor.*—A girl fourteen years old had severe pain and paresthesia of the left upper extremity accompanied by some weakness. Fifteen months later she developed spastic paralysis of the left leg; three months later there was flaccid paresis of the right arm and spastic paresis of the right leg. About the same time she had pain in the middle of the cervical region of the spinal column and tenderness upon pressure over the fourth and fifth vertebræ. In the course of the next six months she developed almost complete spastic paralysis of the legs. There was some atrophy of the arms on both sides and hypalgesia and hypesthesia on the ulnar sides. There was some disturbance of the bladder and rectum. The order in which the symptoms appeared was rather against tumor of the spinal column, and they were rather difficult to make agree with tumor of the cord itself, therefore there was reason to believe that the meninges were involved. It was not possible to decide whether it was extra- or intradural although the early development of the bone symptoms speaks in favor

of the former. These symptoms Meyer believes in agreement with Bruner, are not trustworthy. The segments or roots involved were apparently the fifth, sixth and sexenth, and probably on account of the hypesthesia and hypalgesia on the ulnar sides of the arms, the eight cervical and first dorsal segments. The clinical symptoms were remarkable, partly for the variation from time to time in the areas of disturbed sensation, and the frequent involuntary flexion of the legs. At the autopsy the stomach was found to be enormously dilated. This was possibly the result of the lesion of the spinal cord. A tumor was also found on both sides of the dura, constricted at the point at which it passed through this membrane. It was about three by one cm. in size, shaped something like an egg; its surface was smooth, it was firm in consistency, and directly involved the sixth, seventh and eighth cervical and dorsal segments. It also involved the left side of the cord and pressed from the anterior surface backward. Histologically it proved to be a fibro-sarcoma. Operation was not performed partly because hypertrophic cervical pachymeningitis was suspected, and partly because the condition of the patient was so bad.

14. *Mutiple Sclerosis and Transverse Myelitis.*—Flatau and Koelichen report the case of a woman sixty years of age who had been exposed to cold. After this she developed fever, had pain in the legs, no control of the bladder, and obstinate constipation. She became bedridden, was unable to walk or turn over in bed; there was almost complete paralysis of the right leg, although she was able to flex the thigh, and still had slight power of adduction; there was almost complete paralysis of the left leg, the movements being even less than on the other side. There was slight spasticity of the flexors of the right thigh. Although the legs were emaciated there were no muscular atrophies. There were some slight sensory disturbances, loss of the patellar reflex on the left side and great diminution on the right. Severe irritation of the sole of the foot caused withdrawal of the leg. There was a bedsore over the sacrum and over both trochanters. The patient gradually grew worse and died. At the autopsy nothing was found macroscopically in the brain and spinal cord. A very careful description with illustrations is given of the results of the Kneisel, Weigert and Marchi methods. The changes found could be classified in two groups: first, early degeneration with degeneration of the myelin fibers not involving the blood vessels or neuroglia; and second, advanced areas of degeneration with involvement of the blood vessels and the neuroglia, and the presence of a great number of compound granular cells. The authors regard the case as an atypical form of multiple sclerosis. There were very slight changes in the nerve cells and in the axis cylinders, which they consider characteristic of this disease, and they classify multiple sclerosis therefore as a diffuse process involving inflammation of the blood vessels of the nervous system, especially the spinal column. In an editorial note Strümpell states that he believes the case to be one of acute disseminated myelitis, and not multiple sclerosis.

15. *Cerebral and Cerebrospinal Ataxia.*—Lüthje reports the following three cases of acute ataxia. A boy of ten years was suddenly attacked by headache, prostration and vomiting. There were no mental symptoms for the first two days, when the patient became violently delirious, there were signs of bronchitis in both lungs with a roseola eruption. The fever lasted for about five weeks, gradually falling to normal. The patient throughout the greater portion of the time was unconscious, then became confused, had to be fed, had peculiar twitchings in the muscles of the face. The Widal reaction was positive. A month after the temperature had become normal the patient began to show signs of returning intelligence, and a

month later it was impossible to make a careful examination. At this time, aside from a rather exaggerated emotional state, his intelligence was normal. Speech was deep, somewhat nasal and monotonous, pauses frequently occurred between words and syllables, and the different syllables were sometimes enunciated in an explosive manner. The motor power was not altered. At any attempt at voluntary motion, however, there was extreme ataxia and sometimes associated movements of the other arm when one of the upper extremities was moved. Walking was entirely impossible; sensation was normal; the reflexes were increased; and there was ankle-clonus. The patient gradually improved, was able to sit up alone, the ataxia disappeared to a large extent, although it was more pronounced in the left than in the right arm, and was still evident in the legs. There was bilateral disturbance of the stereognostic sense. Two months after convalescence from the fever, speech was still gravely disturbed. The second case, a sister of the first, nine years old, was brought to the hospital suffering from a severe attack of typhoid fever. She was unconscious for weeks, and five weeks after onset of the disease began to have twitchings in the face and rhythmical lateral movements of the head. During convalescence examination showed that speech was toneless, the syllables were pronounced slowly, occasionally explosively; there was marked ataxia in the movements of the arms and legs, especially the latter; the pattellar reflexes were exaggerated, and there was distinct ankle-clonus. When the patient attempted to stand there were violent oscillations which were not increased by closing the eyes. Sensation was not disturbed. She gradually improved, but when discharged had difficulty in walking, and the speech was still monotonous and scanning. The third case, a six-year-old brother of the two preceding cases, was brought to the hospital suffering from typhoid fever, which also proved to be a severe attack. During the attack he had dilatation of the right pupil, was unconscious, later confused, and also developed rhythmical lateral movements of the head. When excited he had disseminated erythematous eruption over the head and face, exquisite ataxia in the head and trunk, and no control of the sphincters. There was no disturbance of sensation, but the stereognostic sense was not tested. Speech was slow and monotonous and the syllables were separated. The tendon and skin reflexes were exaggerated. This patient rapidly improved and was discharged almost well. The cases are very similar, and Lüthje believes they belong to the pure cerebral form of acute ataxia. In one case, however, the symptoms indicated involvement of the cerebellum. He discusses the differential diagnosis of spinal, bulbar and cortical ataxia and is inclined not to exclude the latter because hemiparesis and hemiplegia did not exist; because the clinical course showed that whatever the lesions were they were exceedingly slight. He believes that the disturbance of the stereognostic sense is one of the most important signs of the cerebral origin of the disease. The disturbance of speech did not indicate bulbar, but rather cortical involvement. He concludes with a careful study of the literature, having found and abstracted sixteen cases, in many respects similar to his own. Some of these were purely cerebral, and a few evidently cerebrospinal in origin. In an annotation he states that four months after the patients were discharged the two younger were almost entirely well; speech was slightly slow, and the patellar reflexes were exaggerated. In the first case the speech was still scanning and monotonous and the reflexes were exaggerated and there was still slight ataxia in the extremities.

16. *Frontal Lobe Tumors.*—Auerbach reports the case of a woman forty-eight years of age who had always suffered from migraine. Usually

she had been industrious, but she began to neglect her work, became irritable, complained of fatigue, had increasing headache, and often refused to leave her bed. From time to time she vomited. The condition gradually grew worse, she ceased to take interest in things, often refusing to eat unless urged. She was apathetic, unclean, and slept considerably. There was some swelling of the face. The nervous system, with the exception of increased patellar reflexes without ankle-clonus or Babinski's phenomenon, and slight tremor of the hands on motion, was negative. Intelligence seemed to be considerably diminished. Memory for recent events was impaired; there was no tendency to joke. There was a suspicion of myxedema; the patient was given thyroid tablets without benefit. She had an apoplectiform attack during which she was unconscious for half an hour, but subsequently recovered, when it was noted that she had difficulty in fixing the eyes, the pupils were dilated, swallowing was difficult and the patient was bent forward and complained of pain in the spinal column. Examination of the eyes showed choked disks. Later she developed stiffness of the neck, and pain upon movement of the head, gradually grew worse, became comatose and died. At the autopsy the frontal lobes were found flattened, the dura was adherent in this region, and the veins were more congested here, especially over the left frontal lobes. The base of both frontal lobes was occupied by a tumor about the size of an apple, extending from the anterior perforated substance forward. The surface of the tumor was somewhat nodular; the mass had about the consistency of liver tissue, and the color varied from pink to dark red. Microscopically it proved to be a fibrosarcoma, probably arising from the dura of the base. The diagnosis presented many difficulties. At first Professor Edinger, who also saw the case, thought of precocious senile dementia, or arteriosclerosis in the vessels of the brain. Later tumor of the frontal lobes was suspected, but the only symptoms were the headache and the psychical disturbances. Of the latter, disturbance of memory and loss of the power of attention, the loss of any initiation or apparently voluntary action, were the most prominent features. Auerbach discusses the theory that intelligence is located in the frontal lobes. He believes that in the human brain they are to be regarded as the most important center of association. Among the interesting symptoms are the apoplectiform attack, the occurrence of choked disks, the absence of disturbance of equilibrium, and the absence of a tendency to make jokes. There is apparently no other case in which the tremor of the hands was so marked. Among the negative symptoms it may be mentioned that the temperature was not elevated at any time, and smell apparently was not disturbed. Toward the last, in addition to the tendency to fall forward, and the pain in the neck, slight paresis of the left facial nerve had developed. Operation was not performed, chiefly on account of the objection of the relatives.

17. *Relapsing Bilateral Ptosis.*—Bychowsky reports the following interesting case. A girl of fifteen years gradually developed ptosis. In the morning she noticed that the eyelids felt heavy, and toward evening she was unable to lift them. The condition lasted three days when electricity was employed and she recovered. At the age of nineteen the ptosis again suddenly recurred. It was complete and was not accompanied by contractions, so that it was easily possible to raise the lids. The eye muscles appeared to be normal; the pupils reacted promptly; the patient's nutrition was good; there were no paralyses of the muscles of articulation, although the speech seemed to be slightly nasal. The patient was given galvanic current and felt better, although the ptosis persisted. Finally, however, during the night it suddenly disappeared. At the age of twenty-one a third attack occurred. On this occasion the patient had severe headache, but the

symptoms otherwise seemed to be the same as in the last attack. The electrical reactions of the muscles of the eye were normal. It was noted, however, that the muscles of the arms became very rapidly fatigued upon exertion, so that after lifting them five or six times they were practically paralyzed. During the application of the electricity to the muscles of the face the ptosis suddenly disappeared and there was not myasthenia of the muscles of the eye; it persisted, however, in the muscles of the arms. In the course of four days, however, this had also vanished. Bychowsky gives a careful discussion of the literature of myasthenia and recurrent paralysis, and reaches the conclusion that his case belongs to the group of myasthenic paralyses. It also bears a certain relation to the Vertige paralysant of Gerlier, and the kubisagari of Miura, probably identical conditions. In a postscript he mentions the fact that at the age of twenty-two the patient had a fourth attack during which Bychowsky was able to determine the presence of the myasthenic reaction in the ocularis and biceps, and the extensors of the fingers on both sides. This reaction persisted for a time and then the patient improved.

18. *Cystic Meningitis.*—Rosenblath reports the following case. A man of forty-seven years was suddenly attacked by trembling of the limbs so that he was obliged to sit down. This continued for two weeks and rendered him incapable of even dressing himself. Later he began to have headache on the left side, was depressed and stopped work. A careful examination failed to show any changes in the motility, sensation, or the reflexes. Finally the patient complained of inward tremor; there was no obvious oscillations of the limbs. Later he developed extremely severe headache and pain and stiffness in the neck. Then there were attacks of vertigo causing him to fall down. It was then observed that he had choked disk. The forehead was tender to percussion, but motility, sensation and the reflexes were normal. There was neither ataxia nor tremor. The attacks of vertigo were sudden, causing him to fall heavily although he rarely lost consciousness. His intelligence gradually altered, his memory was impaired, and occasionally he seemed confused. Sometimes he had hallucinations of vision. His mind gradually failed; he developed agraphia and alexia, became more and more apathetic, and toward the end had vomiting. There were some anesthesia in the right half of the body; his temperature rose, he had a tendency to sleep, and finally died of dyspnea, and paralysis of the muscles of deglutition. At the autopsy some calcareous infiltrated cysts and some clear cysts were found in the neighborhood of the chiasm at the foot of the third frontal convolution on the right side; at the top of the temporal lobe and over the pons and medulla. A large cyst was found also in the posterior portion of the left lateral ventricle. A few cysts were also found in the upper portion of the cervical enlargement. Around these cysts there was occasionally cellular infiltration, particularly in the neighborhood of the blood vessels. A diagnosis of cysticercus racemosus of the brain was made. It is probable that the symptoms were produced rather by the involvement of the meninges and the blood vessels in the neighborhood of the cysts than by the cysts themselves. Rosenblath believes that cysticercus racemosus of the brain is not so very rare; death usually occurs slowly, the symptoms lasting one or two years, although they may last longer; and Zincker has believed that in two of his cases the symptoms persisted for seventeen or eighteen years. The earliest symptoms are those of neurasthenia; later there is weakness of the limbs, pain and stiffness of the neck, then attacks of weakness associated with tremor of the extremities; headache and vertigo are quite common. Cramps have been described in about half of the recorded cases; they may be slight twitchings of the arms

and legs, or they may resemble epilepsy. Not infrequently paresis may occur. The eyes may become prominent, there may be diplopia or ptosis. In many cases there is sudden transient blindness, and in a few hallucinations of vision. Aphasia is rare. Cerebral vomiting is very common. As a general rule patients become demented, but occasionally they are maniacal. It is possible that many of these symptoms are due to vital phenomena on the part of the cysticercus, although it seems to be certain that the majority are dependent upon changes in the blood vessels. It is most difficult to distinguish this condition from cerebral syphilis.

J. Sailer (Philadelphia)

### JOURNAL DE NEUROLOGIE.

(1903, VIII, No. 1, January 5.)

1. Methods of Improving the Organization of the Belgian Asylums. J. Crocq.

1. A discussion of the method of caring for the insane in Belgium and in other countries, with suggestions as to their improvement, which is not suitable for review here.

(1903, VIII, No. 2, January 20.)

1. Facio-scapulo-humeral Hemiatrophy. A. Debray.
2. Case of Hystero Syphilis. Thooris.
3. Note on the Babinski Reflex. C. Heldenbergh.

1. *Facio-scapulo-humeral Hemiatrophy.*—A report of a case of this character beginning subsequent to fracture of the wrist. The author thinks that the long continued sensory irritation due to the wrist injury may have sufficed to determine a functional disturbance in the trophic nerve cells situated in neighboring medullary segments, hence the atrophy. There was paradoxic electric reaction in the atrophied muscles, faradic irritability and,to a less extent, galvanic irritability being increased. There was some improvement under the continued use of faradism with rubbing and suitable diet.

2. *A Case of Hystero Syphilis.*—Report of a case of hemiplegia and hemianesthesia, in a man of twenty-seven years of age, formerly syphilitic, in which the combination of symptoms, and the lack of effect from syphilitic treatment, caused the author to conclude that the trouble was of hysterical, and not of organic origin.

3. *Note on the Babinski Reflex.*—A short discussion of the method of production of this reflex which the author thinks the type of abnormal cutaneous reflexes, an extrapyramidal, or "spino-mesencephalic" reflex, as he calls it.

(1903, VIII, No. 3, February 5.)

1. Multiple Sclerosis. Bouchard.
2. Bilateral Atrophic Paralysis of the Tongue. Decroly

1. *Multiple Sclerosis, Movements During Repose.*—Description of a case observed during six years (from the age of sixteen to that of twenty-two years), which presented the following sequence of events. At first headache, vertigo, drooping of the upper lids and deviation of the right eye outwards, next loss of power, and exaggerated reflexes in the lower limbs, with continuous involuntary movements in them, these later being replaced by similar movements in the upper extremities. Speech then became difficult, of a scanning character, and finally unintelligible, while the legs grew so stiff as to make walking impossible. These symptoms, which took a year for their development, remained stationary for a long

time, and then began an improvement which proceeded to a remarkable degree, to "almost a cure." The patient's mental condition, however, remained defective. The author discussing the diagnosis considers especially Friedrich's disease, cerebellar heredo-ataxia, Parkinson's disease and multiple sclerosis. He concludes that while involuntary movements during repose are rare in the latter disease—a few cases of the sort have been reported—the clinical picture upon the whole corresponds best to that of multiple sclerosis.

2. *Suboccipital Vertebral Disease, with Bilateral Atrophic Paralysis of the Tongue.*—A description of a case of this character which began with pain in the head and neck, paralysis of the left arm and leg next appearing, and extending later to the right arm and leg. The man coming under treatment at this time extension, on the inclined plane, was made with so much improvement that he was able to get about and to work again. The bone disease progressing, however, the head became gradually displayed backward, and the tongue underwent complete atrophy, probably from pressure upon both hypoglossal nerves. Next, in spite of treatment, the paralysis of the limbs again increased, and the patient had become completely bedridden at the time of the report.

(1903, VIII, No. 4, February 20.)

1. Differential Diagnosis of Epilepsy. **Kowalevsky.**
2. Sarcolysis in Muscular Regression. **DeBuck** and **DeMoor.**

1. *Differential Diagnosis of Epilepsy.*—Will be abstracted when completed.

2. *Sarcolysis in Muscular Regression.*—Muscle is composed of two distinct morphological elements. (1) The sarcoplasm or non-differentiated protoplasm, with the nuclei, the nutritional element. (2) The myoplasm or differentiated protoplasm, the functional element.

The first is the one reacting to morbid agents. From the study of numerous preparations the authors are convinced that the muscle fiber plays an active rôle in its own regression, that it tends to return to an embryonic plasmodial, and cellular state, and that in this process the nuclei exercise an active biochemical or hystolitic influence, a phenomenon which they call "sarcolysis." The muscle cells play in this process a phagocytic rôle. These "sarcolytes" they think after repassing the embryonic stage can undergo a metaplasia, being transformed into connective tissue, and in this way a great part of the connective tissue, which replaces atrophied muscular fibers is produced, a "muscular autosclerosis."

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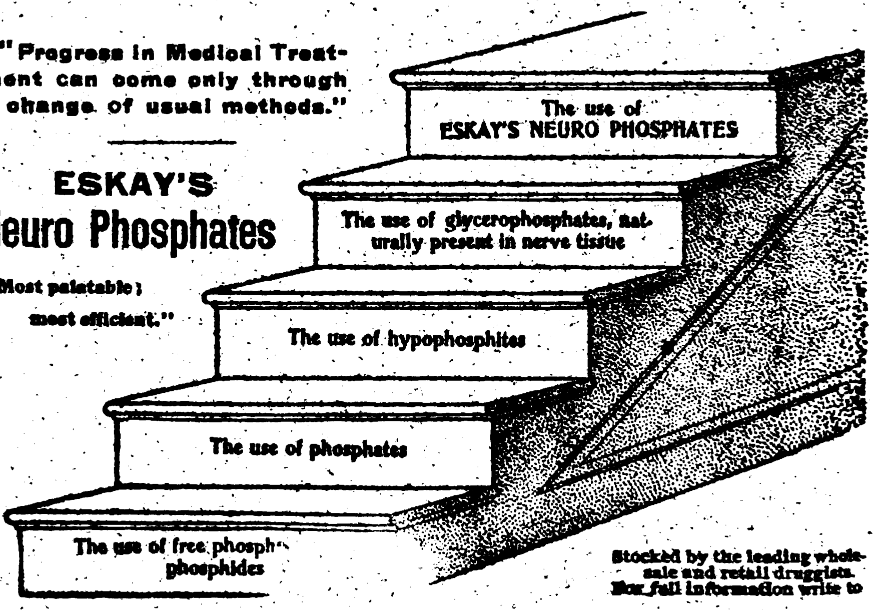
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Vol. 30 October, 1903 No. 10

# The Journal of Nervous and Mental Disease

The Official Organ of

The American Neurological Association
The New York Neurological Society
The Philadelphia Neurological Society and
The Chicago Neurological Society

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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

Printed by the Alliance Press Company, 33 W. 18th Street, N. Y. City

ISSUED MONTHLY $3.00 PER YEAR

FOREIGN SUBSCRIPTION, $3.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, [illegible]

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## Original Articles.

# THE SIGN OF THE ORBICULARIS IN PERIPHERAL FACIAL PARALYSIS.[1]

By Dr. George W. Jacoby.

The divergence of views as to the course of the central neurones of the facial apparatus has always been very great.

All of our knowledge had been derived from pathologico-anatomical, and experimento-physiological observations; clinical study failed to cast any material light upon the subject, because all deductions from clinical observations were dependent upon the assumption that in facial paralysis due to central affections, the tempôro-facial branch of the facial nerve was not involved, and that in ordinary hemiplegia the upper part of the face entirely escapes being paralyzed.

In order to explain this non-involvement of the upper facial, certain older authors questioned whether the upper facial had any cortical origin whatsoever, and believed that its only central source was from the nucleus of the fourth ventricle. Others (Huguenin, Chvostek and Hallopeau), while admitting a cortical origin, assume that the upper facial reaches the facial nucleus from the cortex by means of a course, separate from that of the lower facial; while still other and later observers have assumed that separate centers exist in the cortex for the upper and lower facial, that for the lower facial lying in the lower extremities of the frontal convolutions, while that for the upper facial was variously assumed to lie in the second frontal convolution at its junction with the precentral (Ferrier, Horsley, Hitzig and Duret), or in the inferior parietal lobule (Mendel, Exner and Paneth, and Obersteiner).

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[1] Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

More recent clinical observations have, however, shown that the assumption of non-involvement of the upper facial in hemiplegia is a mistake. The recognition of this long dominant error in clinical knowledge is due in great part to the sign of the orbicularis (Signe de l'orbiculaire de la paupière). This name was first suggested by Révilliod to describe a symptom which was already known but which he above all others has most carefully studied and utilized for scientific deductions.

This symptom consists in the inability of a hemiplegic, voluntarily to close the eye upon the paralyzed side, except in conjunction with the other eye.

Thus in cases of hemiplegia, the temporo-facial branch of the facial nerve is often so slightly affected as to lead to doubt whether the upper facial territory is involved at all.

If in such a case the hemiplegic be asked to close both eyes together, he is able to do so without difficulty; he is also able to close the eye upon the sound side while keeping the eye upon the affected side open, but he is unable to reverse this procedure and close the eye upon the paralyzed side while keeping that upon the unaffected side open.

This sign of the orbicularis was found by Révilliod as well as by subsequent observers, Boiadjiew, and Bard, to be present in almost all hemiplegics, and this not only for a short time after the apoplectic attack, but frequently persisting for a long time after all other signs of paralysis have passed away.

Every subsequent investigator has been able to corroborate these earlier observations, so that as a result of this, and other clinical signs which do not concern us here, it is today universally admitted that the upper facial is involved in all central affections of the facial nerve, although to a less extent than is the lower facial.

This being acknowledged, it is also clear that the various assumptions regarding the dissociate cortical origin of the upper facial are superfluous and the most modern authors agree with Mills who sums up the central course of the facial as follows:

"The central neurones of the facial apparatus arise from the motor cells of the lower extremities of the central convolutions. Together they form a tract which passes by the corona radiata and the internal capsule about its knee to the tegmentum, decussating in the latter to enter the facial nucleus of the other side."

If, however, the implication of the upper facial in the central paralysis is a generally recognized fact, it must be admitted on the other hand also that the upper facial in such paralysis is always relatively free, that it is rarely as much involved as the middle and lower facial or as much as the upper facial is involved in peripheral paralysis.

This fact requires explanation!

Why is the upper facial relatively free in central paralysis?

*A priori,* it may be reasoned, the functions of the upper and lower facial territories differ materially, the upper being utilized more for emotional movements, while the middle and lower serve more for voluntary ones: that emotional movements are as a rule bilateral, while voluntary ones may be either unilateral or bilateral; that, furthermore, inasmuch as the voluntary movements in cerebral hemiplegia due to lesion of the pyramidal tracts, are the ones most affected, it will be comprehensible that the middle and lower facial muscles will be more markedly involved than those supplied by the upper branch of the facial nerve.

This reasoning presupposes a special path, not through the internal capsule, for emotional movements, or it warrants the assumption that each upper facial is innervated from both hemispheres. While the first assumption is by many considered correct, it would, however, in the case of cortical or subcortical lesions (that is, involvement of the purely voluntary motor paths), necessitate equable paralysis of all parts of the facial, which is contrary to all clinical experience.

The other assumption was first advanced in a general way by Broadbent, and is almost universally accepted as a fact.

Broadbent's hypothesis in the main represents the following ideas: Many muscles of the body are used only in conjunction with their fellows of the opposite side; others while usually used alone may be and frequently are used conjointly with their fellows. Those muscles which are always used alone are represented only unilaterally in the cortex; those that are used conjointly are bilaterally represented, so that unilateral movements can be excited only from one, the opposite hemisphere, while bilateral movements can be excited from either hemisphere, and finally those muscles which are sometimes used alone and at other times conjointly are connected with both hemispheres, but are generally excited from one, the opposite hemisphere, only.

The synergic action of the orbiculares palpebrarum, during repose or during a state of unconsciousness, shows that they must have a bilateral innervation and that they must, therefore, be excitable together from either hemisphere. As a result of education and under influence of the will each orbicularis may in its action become independent of the other, and must then be excitable individually from the contralateral hemisphere. Such conjoint and independent actions can be explained only by the assumption of a chiasm formation, an interlacement composed of two crossed and two direct tracts. Therefore Révilliod, starting from the hitherto accepted premise that the sign of the orbicularis is never present in peripheral paralysis, but is found only in central affection, seeks this chiasm formation at a point on the motor path between the cortex and the nucleus. Révilliod says: "If indeed, as the sign of the orbicularis has demonstrated, the superior facial is really implicated in ordinary hemiplegia together with and by means of the same focus as the lower facial and the extremities, then it must form part of the same tract, called pyramidal, and course with it from the cortex to the oblongata, passing by way of the internal capsule. But in order to assure the synergy of the two sides of the upper face, this nerve must divide before reaching its nucleus, into two branches, the one crossed, the other direct, the latter returning to the face upon the same side as the hemisphere from which it emanates," and "the crossed branch would consequently always be accompanied and assisted into its end ramifications, by the direct branch which comes from the hemisphere of the opposite side." This explanation Révilliod illustrates by a diagram of the motor tract in which the decussation of nerve fibers is shown to be complete for the lower facial (and the extremities) but is assumed to be incomplete for the synergic muscular groups of which the orbiculares form a morphological part.

Révilliod's assumption may be endorsed but at the same time may be considered insufficient.

If we recall that the upper facial also in other pathological conditions than that of central hemiplegia may show a comportment different from that of the middle and lower distributions; that in bulbar paralysis it is almost the rule to find the upper facial relatively less implicated than the lower facial, and that even in purely peripheral facial paralysis the temporo-facial branch is not infrequently much less affected than is the lower facial, then we are entitled to assume, not only as has been done by others, that the upper facial is especially resistant to pathological influences, but also that a decussation of fibers must take place at the facial nucleus itself and that this decussation is mainly made up of those fibers which act synergically.

This question of decussation of facial fibers is one which anatomical and physiological investigations, as well as pathological studies, have been unable to answer definitely.

*A priori,* and reasoning from analogy it would seem that such decussation must exist. Clinically we know that in complete hemiplegia, not only is, as has already been emphasized, the orbicularis palpebrarum relatively free, but also that the eye muscles, the muscles of the trunk and of the larynx, in short all those muscles which usually functionate in pairs and give rise to bilateral associated movements are comparatively unaffected.

This can be explained only if we admit the existence of commissures joining the nuclei of origin of the motor nerves of the two sides, such commissure then assuring functional physiological synergy, and reciprocal subserviency in pathological implication of the nucleus upon one side.

As a general principle the existence of such decussation between the homologous nuclei of all motor cranial nerves is admitted (see Edinger), but it is not admitted as a fact by all observers for all cranial nerves.

Thus as regards the facial, we find that Adolf Meyer, C. Mayer, Van Gehuchten and Bischoff have furnished proof in opposition to the doctrine of decussation between its roots.

On the other hand, Obersteiner, von Bechterew, Flatau, Marinesco, Bruce, Kotelewsky, Mills, Pardo and Bary are supporters and have furnished anatomical proof of the existence of such decussation.

Recent observations of Ramon y Cajal are also in accord herewith and clearly show that fibers belonging to the seventh nerve decussate in the raphè.

Of special interest and value in this connection, are the experiments of J. and N. Aspisow. These observers after having, by means of electric excitation, located the cortical center for the upper facial in dogs, excised this center and after proper lapse of time examined the brain.

In addition to secondary degeneration through the corona radiata, internal capsule, crus and pons, the degenerated fibers could be plainly traced from the homolateral, across the raphè, to the contralateral facial nucleus.

Against all this anatomical proof, however, stood the clinically accepted fact that the sign of the orbicularis was absent in peripheral paralysis. As a result of my examination of all the cases of peripheral facial paralysis which I have seen in hospital and private practice, since the last four years, I am able now to say that this so-called fact is an error, and that the sign of the orbicularis can be found in many peripheral cases if sought for at the proper time.

Already in 1894 Mann, in calling attention to the fact, which had also been recorded by a number of others, that the orbicularis oculi in some cases of peripheral paralysis is not nearly so much involved as the other facial muscles, and that this same muscle in cases which are improving shows an early regeneration, also states that in these same cases the patients were able to close the eyes at a time when the attempt to corrugate the forehead failed; the forehead upon the paralyzed side remaining smooth but the eye closing.

If this investigation be probed further, it will be found that while many such patients can at this time close both eyes together, the independent closure of each eye is possible only for the eye upon the unaffected side.

Before this test can be utilized for deductions or conclusions of any kind, it must, however, be first shown that the independent closure of each eye exists physiologically in the majority of individuals. This has been demonstrated by Boiadjiew, who tested

750 healthy persons, and found that 484 or 64.54 per cent could close each eye independently; 126, or 16.80 per cent, could close only the left eye without at the same time closing the right; 64, or 8.53 per cent, could close only the right eye without at the same time closing the left; 76, or 10.13 per cent, could close neither the left nor the right eye independently of the other.

This investigation, therefore, shows that two-thirds could close either eye independently and that, therefore, this capability is sufficiently frequent to allow its absence to be utilized as a clinical symptom, provided the patient can give a history of previous independent control.

There is no necessity for giving a large number of cases in order to demonstrate the frequent occurrences of a symptom which any observer may easily corroborate. Let us take a few typical cases which present points of special interest.

Thus, W. W. O., male, aged thirty-seven, was seen on October 16, 1899; he had then been afflicted with a peripheral paralysis of the right facial nerve since a few days. There was marked lagophthalmus, with inability to corrugate the forehead, complete R. D. in all the muscles supplied by the facial nerve, including the orbicularis oculi; no disorder of taste nor of hearing. Ten days after onset of the paralysis, with R. D. still existing in all the facial muscles, including the orbicularis, and while still unable to corrugate his forehead, to whistle or to blow out a light, he could close both eyes together, but could not close the right eye independently of the left. He could close the eye upon the sound side, alone; he could close the affected eye when the other eye was closed, but he could not close the affected eye alone when the unaffected eye was kept open.

Two weeks later he was able to close each eye freely and independently of the other, as he had always been able to do prior to the onset of the paralysis.

The following case shows the long persistence of an untreated facial paralysis, the improvement comparatively soon after treatment was instituted and the supervention of the sign of the orbicularis contemporaneously with the onset of improvement.

A. L., a young boy, was operated for mastoiditis; after coming out of the narcosis it was noted that he had a complete left-sided facial paralysis. For one and one-half years he was subjected to much treatment on account of his ear trouble, but no attention was given to the facial paralysis. At the time when I first saw him he still showed complete paralysis of the entire facial with marked R. D. in all the muscles supplied by this nerve, no faradic response being obtainable in a single muscle. After five months' electrical treatment, faradic reactions set in in the temporo-facial as well as in the lower facial, but not so marked here as there; at the same time functional improvement took place in the upper facial alone and he was soon able to close the eye completely. He could, however, not close the affected eye without also closing the sound one.

Four weeks later, while still showing R. D. in the entire upper and lower facial territory, he was able freely to close each eye separately.

The mother states that prior to the facial involvement he was able to wink each eye alone.

The last case which I will briefly relate is of interest because it was a case of peripheral facial paralysis with apparent freedom of the orbicularis, which, however, through the presence of the sign, was shown to be an ordinary case with but slight involvement of the upper branch.

The patient, a middle-aged woman, was seen in March, 1900, three days after the acute onset of a right-sided facial paralysis. Examination showed marked involvement of the lower facial, as shown by the interference with mouth movements (whistling, blowing, grinning, etc.), with apparent freedom of the upper facial. She was able to close both eyes completely and easily. When each eye was tested separately it was, however, found that she could close the left eye alone with ease, but that she could not close the right at all except in conjunction with the other eye.

She made a rapid and complete recovery and was then able to close each eye singly as she had been able to do prior to the onset of the paralysis.

Without citing any more cases I desire to call attention to a result of my investigation which seems to me to bear some prognostic significance. As well as it has been recognized that in peripheral paralysis of the facial nerve, the orbiculo-frontal branches are frequently less implicated or recuperate earlier than the other branches, so also it is well known that marked spasm and twitchings may occur in the orbiculo-frontal territory, at a time when the voluntary path is being reëstablished, and at a time in which reduced electrical excitation still exists. In what percentage of cases such spasms occur, I do not know, but the number is probably not small; I am, however, able to state with certainty that in no case which I have followed during the last four years has such spasm occurred after the sign of the orbicularis, which had been present, had passed away; or I may say in all cases in which the spasm has occurred the patient was not able to close each eye independently at the time of the onset of the spasm.

In conclusion I would summarize the result of my studies as follows:

1. The statement of Bard, that the sign is invariably lacking in peripheral facial paralysis and is, therefore, a distinguishing mark of central affection, is incorrect.

2. The presence of the sign in peripheral paralysis is further proof of the existence of commissural fibers between the nuclei of the 7th nerves.

3. The sign is of clinical value in so far as its presence in peripheral paralysis shows that complete recovery has not yet occurred.

4. The secondary over-action in the orbicularis palpebrarum, which is late in appearance and always coincides with some recovery in power, does not occur in those cases in which the sign having been present has passed away.

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# GIGANTISM AND LEONTIASIS OSSEA, WITH REPORT OF THE CASE OF THE GIANT WILKINS.

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(*Continued from page* 532.)

In view of the findings in this case one must admire the keenness of Sternberg's judgment in excluding acromegaly and pointing out the analogy with Buhl's case (to be related later). The relation of acromegaly to gigantism, and of both to disease of the hypophysis, has been extensively discussed of late. Thanks to this fact a number of exhaustive reviews of the cases of all more or less scientifically studied giants exists. The most thorough ones are those by Sternberg (43), Meige (31), and Woods Hutchinson (27). All authors agree that giants frequently present acromegalic symptoms, and that hypophysis disease is the rule in acromegaly. A most daring generalization is that of Massolongo (30), who says: "Acromegaly is nothing but delayed, abnormal gigantism." Meige quotes and indorses this statement and gives the easy explanation that, when the disease commences in youth we get a case of gigantism; when in adult life, acromegaly; if commencing in youth and continuing into adult life we get a combination of the two. After an extensive review of cases* he formulates the following statements (*loc. cit.* p. 458): "1. Acromegaly never precedes gigantism. 2. Acromegaly sets in during the course of about one-half of the cases of gigantism. 3. When acromegaly is associated with gigantism, the latter always appears first." Woods Hutchinson sums up the result of his studies of gigantism as follows: "1. The greater part of the overgrowth is found at or near the tips of the segment-crescents, as in acromegaly, differing from the latter mainly in that it is not exclusively confined to the tip of the segment or last division of the limb.

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*Among these our Wilkins appears as two, the author having failed to observe that Dana and Lamberg described the same man.

2. The facial part of the skull is enlarged out of all proportion to the cranial, particularly in the region of the lower jaw. 3. The condition, whether it be regarded as normal or morbid, is one that distinctly tends to shortness of life, and would appear to have an average duration of scarcely more than twenty years. 4. The mental and physical vigor of the giant is distinctly below par, and his death usually comes either from a steady progressive increase of this weakness, or from some trifling accident, or usually mild, intercurrent disease. 5. Sexual powers appear in the great majority of cases to be far below normal. 6. There is a decided preponderance of males among the victims of this condition; in all of which statements there is a decided parallelism with acromegaly. Last of all, and from the point of view of this essay, of greatest interest, is the fact that the one morbid condition which is peculiar to both of these disturbances of nutrition, the enlargement of the pituitary body, is found to be present in a large majority of cases of both." After stating that the material as yet is meager, he says that we are "justified at least in the tentative conclusion, until some evidence to the contrary can be adduced, that acromegaly and gigantism are simply different expressions of one and the same morbid condition; in other words, that acromegaly is a general overgrowth tendency which does not, for some reason, begin to express itself until after adult stature has been reached, and which consequently expends itself upon those points in the body at which growth last ceased—the extremities of the segment-crescents and the distal extremities of the appendages. Second, that gigantism in a large majority of cases is this same condition manifesting itself in childhood or before complete stature has been reached, and the growth in consequence is more symmetrical and less strictly confined to the last segment of the arches and appendages. In most cases, however, the tendency appears to be for these last segments to grow in an unsymmetrical and excessive manner." After a discussion of the conditions found in the hypophyses of acromegalics, giants and dwarfs, he further concludes: "1. That the pituitary body is still functional. 2. That disturbances of its metabolism are the principal factors in both acromegaly and gigantism, the difference between the results being simply due to the stage of individual development at which the disturbance of the function begins. 3. That the nature of the

overgrowth in both these diseases is primarily on the order of a pure functional hypertrophy; later, however, losing some of the definiteness of its impulse, and either producing immature tissue of a mixed type or resulting in simple hemorrhagic exudation, with either cyst formation or complete breaking down of the tissue mass. 4. That it seems probable, although upon this head the evidence is still uncertain, that some part is played by this body in 'dwarfism,' rickets, and the dwarf forms of cretinism. 5. That a reflex disturbance of its function may possibly underlie the dystrophy accompanying pharyngeal adenoids. 6. That it would appear to be a sort of 'growth center' or proportion-regulator of the entire appendicular skeleton." The author finally hints that chorea may be due to disturbance of the hypophysis, and promises a further report on the subject.

Sternberg, (43) states that about 20 per cent of all acromegalics are over 177 cm. (5 ft. 10 in.) tall, and that about 40 per cent of all giants have acromegaly, but denies the identity of the two processes which up to that time had been advocated by Klebs, Massolongo, Brissaud and Meige. He believes there are "normal" giants, i.e., well-proportioned over-grown people. As such he regards Virchow's case, Winkelmeyer (48), whose height is given as 227.8 cm. Hutchinson does not consider him "normal," as his height at the hip line, as determined by the position of his hand in the picture, is 64 per cent of the total height, instead of the normal 55 per cent. Sternberg classifies the "pathological" giants as follows: "1. Acromegalics—about 40 per cent of all giants. 2. Cases with multiple tumor-like exostoses (leontiasis ossea and hyperostoses). Often the signs of compression of the brain and cranial nerves. Here he groups Buhl's case, Hasler (10), 227 cm.; Sirena's (42) first case, 240 cm., and our case, Wilkins, 245 cm. (Reference given to Lamberg's report). 3. Cases with facial hemihypertrophy. Dana's case.* 4. Cases with multiple curvatures of the bones, as scoliosis, curved humerus, genu valgum, of similar appearances as in rickets; for instance, Bollinger's giantess (6) and Zitterland's case, Berlin skeleton 3040 (51). 5. Gigantism in hereditary syphilis, case of Fuchs (18). 6. Precocious development and rapid growth in cases of testicular tumors in children. Sacchi's case (38)."

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*I suspect Wilkins is the case referred to. Reference is given to the article in which his case is reported.

Meige, who quotes this classification, finds it a little artificial. This may be true, but it probably goes into generalizations as far as the facts warrant at present.

Sternberg sees two possibilities for an etiologic relationship between gigantism and acromegaly. First, the latter may induce a cartilaginous proliferation in a still ununited epiphysis; or, secondly, in gigantism a disposition to general dystrophies, and particularly to acromegaly, may exist.

Schmidt (41) inclines to the latter view, and considers the acromegalic features as something complicating the gigantism, the same being true of the frequent scolioses, genua valga, etc., and the rarer hyperostoses of the bones of the head, as seen in our case, which is, together with that of Buhl, quoted as one in point (from Lamberg's report).

As Buhl's case (10) is in many respects similar to our own, it deserves special mention. It was that of a man, Thomas Hasler, who was normal up to the age of nine years, when a rapid growth commenced. This is said to have followed a kick on the cheek by a horse. Some years later huge hyperostoses of the facial and cranial bones appeared and were larger on the injured side. At twelve he measured six feet. He died at twenty-five. His height then was 227 cm., or 235 cm. (7 ft. 8½ in.) with the back stretched; the weight, 155 kg. There were no lesions of the viscera. All epiphyses, including those of the trochanters, were sharply outlined. The skull showed enormous exostoses. The lower jaw was enlarged in all directions, very irregular, its height at the middle of the chin 12 cm.; thickness, 9 cm.; the left side was the thicker. The upper maxillæ were also thick. On the left side the processus frontalis, the nasal, malar, ethmoid and lachrymal bones were greatly thickened, the floor of the orbit raised, the left wall of the nose displaced to the right. The left frontal bone measured 6 cm. (2⅜ inches) in thickness, the left temporal, 1.5 cm.; the right, 1 cm. The left side of the occipital and parietal bones also thick. Both in the frontal and parietal bones superficial porous islands were found. The interior of the skull was diminished on the left side, the sella turcica displaced to the right. The brain weighed 1,465 grams, and was much decomposed at the time of the autopsy. The hypophysis is not mentioned. Buhl attributed the patient's death to compression of the brain.*

Hutchinson's hypothesis that the hypophysis is a "growth center or proportion-regulator of the entire appendicular skeleton" is a bold one, considering how few actual facts we possess concerning the functions of that organ. Benda in quoting the above statement (4) says that Hutchinson is bolder than himself. The physiologists seem unable to agree about a single function of the hypophysis, and some deny its having any function at all. The studies of Andriezen (2) on the larval amphioxus, ammacetes, balanoglossus and other lower animals are very fascinating.

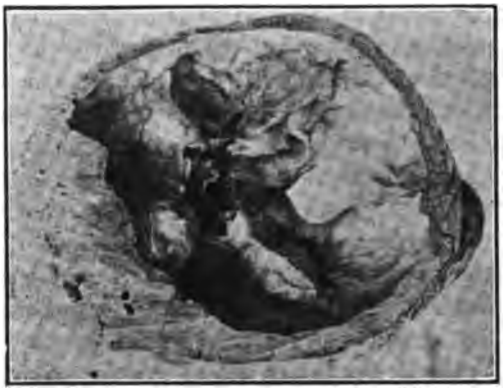

Fig. 10. Base of skull in Buhl's case.

He observed carmine solutions passing from the buccal to the neural cavity through the hypophysis and infundibulum. He says: "A water-vascular system permeating the body-tissues, and serving for the in-bringing of oxygen and the out-taking of waste products, and having inlets and outlets communicating with the aqueous medium in which the animal lives, preceded, in point of time, the development and elaboration of a true blood-vascular system." He believes that the anterior or glandular portion of the hypophysis in these embryos secretes a substance which modifies the fluid passing in from the buccal cavity, while the posterior or nervous portion controls that secretion. His predictions of the symptoms following extirpation are also interesting, as they were confirmed by the experiments of Vasalle and Sacchi (47).

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*A glance at the picture of the skull will show the close resemblance to our case. As the original communication is difficult of access this drawing is reproduced here.

Cyon has performed many experiments and written a number of articles on the physiology of the hypophysis. In the most recent one (12) he relates how he irritated the organ in various ways through an opening made in the pharynx. He claims the following functions for it: 1. (a) It serves to regulate intracranial blood pressure, (b) regulates metabolism. 2. It regulates cerebral pressure in two ways, (a) mechanically, each increase in pressure irritating it, causing slower and stronger heart beats and increased rapidity of venous circulation, particularly of the thyroid, thus relieving the brain; (b) chemically, as it produces substances, probably two in number, one of which irritates the vagus, the other the accelerators. 3. Effect on metabolism takes place through similar action of its products on the vagus and the sympathetic, seen in increased oxidation and decreased weight. 4. Tonic excitation of the vagus depends on excitation of the hypophysis due to pressure exerted on it. 5. Continued excitation, especially electric, of the hypophysis, causes as after-effects severe epileptiform spasms, most easily explained by circulatory disturbances in certain parts of the brain. In rabbits electric irritation caused persistent erections and increased diuresis; the latter also followed the injection of extract of the hypophysis. Cyon believes that the effect upon the circulation is due to a stimulation of thyroid activity, regulating the circulation through the carotids. Hypophysin, the most important substance obtained from the hypophysis, has an action similar to, but more intense than that of iodothyrin. In thyroid disease the hypophysis may for some time take up the function of the thyroid. Thus, enlarged hypophyses were found in thyroidectomied rabbits, and the roof of the hypophysis cavity often ossified, which, according to Cyon's theory, ought to facilitate the function of the gland. But in spite of his conviction of the correctness of his views, Cyon is unable to explain acromegaly either by hyper—or hypo—function. Certain symptoms, such as impotence in men and amenorrhea in women, he interprets as due to a loss of function.

Enlargement of the hypophysis has been described as following thyroidectomy by Hofmeister (26), Stieda (45), Gley (19), Goldberg (20), and others; in cretinism by Bourneville and Bricon (7), Dolega (14), Osler (36), Grön (21), and others. Le Count (32) states that in acromegaly the thyroid has been recorded as reduced in size four times, as cystic or the seat of colloid degeneration, twice, and as normal in appearance five times. Ponfick (37) reports two cases of myxedema in which the glandular atrophy and increase in fibrous tissue were so much more marked in the hypophysis than in the thyroid that it seemed probable the change had begun in the former organ, which Ponfick suspects may be closely related to the production of myxedema.

Friedman and Maas (17) disagree with Cyon's views, and from a number of extirpations of the hypophysis conclude that it is not essential to life, and state that, in their experiments, its absence led to no change in any organ, particularly not in the thyroid.

Castelli (11) gives as the effects of extirpation: More rapid heart action, slower respirations, psychic depression, tetanic-clonic cramps, and cachexia, ending in coma and death. He believes it is concerned in the rendering harmless of toxines arising in the organism—which is, in turn, denied by Cyon.

Biedl and Reiner (5) also attacked Cyon's views, and claim that electric irritation of most places of the brain has the same effect as when applied to the hypophysis.

With such lack of definite knowledge as to its functions, it is no wonder that the rôle played by the hypophysis in disease can only be guessed at for the present. No one can deny the fact that it is diseased in most cases of acromegaly and in many giants, but whether this is the cause or merely itself a phenomenon of the disease cannot be stated. It is also probably true that the majority of the hypophyses tumors observed in acromegaly are to be considered as hyperplasia of the glandular portion. They have been given a great variety of names, but as Benda (4) well puts it, the names given differ more than the descriptions, and the histological picture in a given case may vary so much that one may find microscopic fields to fit almost any tumor name. In four such cases Benda succeeded in demonstrating glandular structures and transitional forms between these and the irregular sarcoma-like portions found. In the present case it was confidently expected to find such transitional forms, and not until the serial sections through the hypophysis had demonstrated the existence of a sharp border at every point, was the glandular origin of the tumor definitely abandoned.

Many cases of hypophysis disease without acromegaly or other disturbance of growth are on record, as Handford's case (22), Woolcombe's case of psammoma (49), Beadle's two cases of gumma and tubercle respectively (3), Breitner's of adenoma (8). In this country Hektoen (23) has reported a case of gumma associated with a thick skull, Stewart (44) and Hinsdale (25) of sarcoma with no acromegalic symptoms. Recently Taylor and Waterman (46) reported a case of a sarcoma involving the optic commissure, but its connection with the hypophysis was not proven, and there is no mention of an examination of the sella turcica after the removal of the brain. In an autopsy on a demented female epileptic, aged 38 years, at the Iowa State Hospital, Mount Pleasant, the writer found an hypophysis that weighed 59 grams. The skull, particularly the frontal bone, was thick. The brain weighed 1,040 grams. The woman was very small and had no acromegalic symptoms. In an insane, tuberculous subject, likewise with no acromegalic features, a small calcareous nodule was found in an otherwise normal hypophysis.

The following instance of gigantism, associated with a tumor of the pineal body, while the hypophysis was normal, is of considerable interest. It is reported by Oestreich and Slawyk (35). A male child had been delivered by forceps and been long asphyxiated. There were spasms of the glottis at the age of three months. The child developed normally the first three years, then it became morose and the body began to grow rapidly, particularly the penis. Walking became difficult. July 4, 1898, four years old, it was admitted to the Berlin Charité. Height 108 cm., weight 20 kg., muscles and skeleton powerful. Its size was that of a normal child of seven or eight years. Skin normal. Head symmetrical, circumference 53 cm. (4 cm. above normal, according to the authors). There were sluggish reaction of the pupils to light, slight convergent strabismus, and a bilateral choked disc.

Sight appeared impaired. Mammæ, hypertrophic; penis, 9 cm. long; pubic hair, 1 cm long. Extremities of natural length. Sensations normal. After three weeks of clonic convulsions the child died Aug. 12, 1899. An autopsy was held. Anatomic diagnosis: 1. Cystic psammo-sarcoma of the pineal gland. 2. Internal hydrocephalus. 3. Gigantism. The skull was irregularly thinned. The posterior part of the third ventricle was occupied by an irregular, partly cystic tumor, the size of a small apple, in the position of the pineal body. The tumor also shows firmer parts and sand-like areas. The thyroid and hypophysis were normal, the thymus of proportionate size and normal histologically. The tumor was found to be a spindle-celled sarcoma, and the authors consider it the cause of the giant growth.

In spite of the title of the article, "Gigantism and Tumor of the Pineal Gland," and the explicit statement that the hypophysis was normal, Meige, in his previously quoted article (p. 472), in referring to this case, says that Oestreich and Slawyk described an acromegalic giant in whose *hypophysis* a tumor was found!

The presence of calcified patches in the spinal meninges of our case is of some interest. The deposit of externally generally smooth, internally rough flattened plates of bone on the inner surface of the dura has been designated "arachnitis ossificans" (40). According to Zanda (50) the bone formation takes place from the dura after adhesions with the arachnoid have been formed. They are most frequent in connection with regressive changes, such as those of old age, chronic insanities, and diseases of the spinal cord. Sainton and State (39) describe a case of acromegaly in a man 54 years old, with numerous such sand-paper-like patches enveloping the cord in the thoracic and lumbar regions, and considered them the cause of the cramps and pains in the extremities present. Similarly located bony or bone-like patches in acromegalics have also been described by Duchesneau (15), Henriot (24), and Finzi (16). In our case, it will be remembered, calcareous nodules were also present in the pleura and peribronchial lymph glands. Von Moraczewski (33) has shown a tendency to retention of lime and phosphorus in acromegalics, and Meige sees here a chance for a new proof of the identity of acromegaly and gigantism, if such a condition is found in the latter, as we might then presume that in the growing giant the osteogenetic function is exercised only at the epiphyseal junctions, while, after the cessation of growth, the osseous hyper-function leads to the formation of aberrant calcareous deposits.

Heredity does not seem to play a significant part in the etiology of gigantism. In most cases the other members of the family are reported of ordinary stature and in good health. Thus "Lady Aama," the French giantess reported by Hutchinson (28), was the youngest of fifteen children, all the others being normal. If the sources of information of De Neuville (34) were even approximately reliable, a tendency to giant growth has existed in certain families. According to him, Robert Hales, an Englishman born in 1820, measured 7 ft. 6 in., his father 6 ft. 6 in., and his mother 6 ft. An ancestor who lived at the time of Henry VIII measured 8 ft. 8 in. Five sisters averaged 6 ft. 3 in.; the tallest, 7 ft. 2 in., died at 20 years of age. Four brothers averaged 6 ft. 6 in. Hales himself measured 62. in around the chest, 64 in. around the waist. Louis Frenz, born 1800, measured 7 ft. 6 in. He said one sister was "nearly as tall," and one brother "still taller." Achard and Loeper (1) report the case of a male acromegalic giant, height 212 cm. (6 ft. 11 in.) whose father measured 195 cm., paternal uncle 210 cm., and a sister 180 cm. According to Meige, the Chinese giant Chang, himself 8 ft. tall, said he had a sister 8 ft. 4 in.

Nearly all giants have been poorly developed sexually. With the possible exception of Winkelmeyer (Virchow's "normal" giant) none have left children, and all of them have been short-lived. Hutchinson gives the average age at death in eight giants as 21.3 years, the extremes being "in childhood" (the tall girl of Basle) and 25 years (Buhl's case, Hasler). According to Meige, the Chinese giant, Chang, must have reached 51 years.

The changes in the skull in our case are difficult to understand, partly on account of the complicated condition, partly because of the small number of analogous cases and the incomplete descriptions to be found of them. While there are many good gross descriptions and accurate measurements, histologic examinations have rarely been made. An attempt will be made here to review briefly the more important cases of cranial hyperostosis which are likely, at least in part, to be of a similar nature.

In 1697 Malpighi (69) described a skull weighing 3,618 grams, with effaced sutures, a tumor of cherry size near the saggital suture, and here and there small marble-like eminences. The temporal bones were particularly large, the base of the skull thick, the orbits small.

Tarin (89) in 1753 described a thickened frontal bone with prominent angular processes.

Jourdain (67) and later Ribel (77) described the case of Forcade, the son of a French surgeon. He was well up to the age of 12 years when an abscess at the inner canthus of the right eye was opened and discharged for a long time. During this time an almond-sized prominence appeared on the nasal process of the right superior maxilla, and increased in size so that at the age of 15 years the boy was unable to breathe through the nose on account of it. Then, the lower jaw, nasal and malar bones, and floor and sides of the orbit became involved. Later, exophthalmos, blindness, and speech troubles appeared. The patient, who was below middle stature, died at the age of 45 years. The dried skull weighed 8 lbs. 3 oz., the lower jaw alone 3½ lbs. There were huge exostoses of ivory hardness on the lower jaw and on the inferior margin of the orbits. The bones of the cranial vault were thick, with flat exostoses and very hard, the frontal and maxillary sinuses obliterated; cranial cavity not diminished in size. The facial muscles were fibrous in appearance and adherent to the periosteum. The bones outside the skull were unusually delicate.

Sandifort (78) described a skull thickened by about one inch with abundant spongy substance between the two thickened tables. On the interior, especially of the frontal bone, were numerous rough projections.

Jadelot (66) described and recognized as pathologic a skull which a previous writer (Wendelstedt) had contended must have belonged to a preadamic giant, at least 13 ft. tall, and which Cuvier, from the condition of the teeth and sockets, in turn had shown to have been that of a child of six or seven years. It weighed 3,913 grams, measured 236.9 mm. in length, 151.1 mm. in height, and 203 mm. in biparietal diameter. The thickness varied from 19.8 to 40.2 mm.; the diploë was more compact than the outer and inner plates.

Saucerrotte (79) describes a man, 39 years old, the circumference of whose head was 797 mm.; the lower jaw, the hands, feet, spine, ribs, sternum and clavicles were large, with hyperostoses. The lips were thick. The position of this case is somewhat uncertain. Virchow, Paget, and Marie each claimed it for the diseases named by them, leontiasis ossea, ostitis deformans, and acromegaly respectively. Ilg (65) and later Gruber (61) report a case whose skull is the now famous one of the Prague Anatomic Museum. A girl of ten years, previously healthy, was taken with amaurosis, followed by severe headaches, delirium and epileptiform attacks. At sixteen she became deaf, at seventeen unable to swallow solid food, lost the sense of smell, and became bed-ridden. During this time she had several attacks of erysipelas. She died in 1804, 27 years old. The dry skull without the lower jaw, teeth and nasal bones weighed 4,200 grams. The fronto-occipital diameter was 197.6 mm., the biparietal 171.2 mm., the thickness varied from 19.8 to 52.6 mm., the occipital bone being the thickest. The external surface was uneven, the sutures had mostly disappeared, the prominences, particularly the external occipital protuberance, were enlarged. There were compact exostoses on the inner side of the frontal bone and greater wing of the sphenoid. The sella turcica was unusually shallow and narrow, and all foramina diminished in size. Diploë was only present in the frontal and parietal bones, in other places replaced by hard bone. The capacity of the cranial cavity was 1080 c.c. The specific gravity of the bone was 1.94. Microscopic examination: "The medullary canals narrow, surrounded by concentric bony lamella, mostly without sharp borders, the peripheral lamella darker than normal. Ground substance fibrillar, darker, with numerous powder-like dark granules. Bone corpuscles not clearly outlined, globular, or at least more globular than spindle-shaped; there are also some abnormally large spindle-shaped corpuscles. The ductuli chalicophori increased in size and number, with fewer anastomoses than normally." This is probably the first histologic examination ever made of a hyperostotic skull.

A very thick skull in Spurzheim's collection was described by Kilian (68). Its thickness was from 27 to 54.1 mm., latter in parietal region.

A still thicker one was found near Münster and recorded by Bojanus (54). Its weight without the lower jaw was 4,000 grams, its thickness 27-67 mm. Most sutures were obliterated, no diploë present. The orbits narrow, their walls thickened.

Breschet (55) reports a congenital case in a child who died at the age of 18 months in a tetanic spasm after having suffered with convulsions from birth, with only brief intermissions. All the cranial bones, except at the base, were thickened, in places measuring one inch. The anterior fontanelle had not disappeared. Bones of the face not involved.

The skull in Vrolick's (91) case weighed 1,305 grams, measured 199 mm. in length, 150 mm. in width, rough externally. The sutures, with the exception of the squamous, were obliterated. The frontal bone was 15-20 mm. thick, occipital 20 mm., right parietal 7 mm., left 15 mm., sella turcica narrow and deep.

In 1851 Albers appeared with a monograph on the subject (52), in which he denied the inflammatory nature of cranial hyperostosis and any connection with rickets, considering the condition one of hypertrophy with deposit of lime salts.

Schützemberger's case (81) is thought by Baumgarten to bear resemblance to the Ilg-Gruber case. The patient died in convulsions at forty years after having suffered from headaches, epileptiform attacks, and mental enfeeblement for some years. The skull was thickened and hard, diploë and sutures absent. The cavity was small, thickening being mainly at the expense of the inner table. The foramina were slightly diminished in size.

Huschke's case (64) was that of a woman seventeen years old whose skull weighed 3,224 grams and measured 228 mm. in length and 157 mm. in width. The thickness of the frontal bone was 10-35 mm., of parietal 22-35 mm., of occipital 16-40 mm. The diploë was mostly replaced by hard bone. The author considered the lesion due to rickets, a view which has been proven untenable by Virchow, Schmidt and others.

Gaddi (60) described a skull found in 1862. It weighed 3,660 grams. Surface rough, sutures obliterated. The bones of the face very large, the superior maxillæ particularly enormous. The lower jaw weighed 422 grams. The only other bone found, the second cervical vertebra, was also enlarged.

The first thorough sifting and elaboration of this accumulated material was made by Virchow, who also proposed the name leontiasis ossea (90), which was suggested to him by the leonine appearance, in the case of Forcade in particular. He expressed the opinion that the process corresponds to elephantiasis of the soft parts, and that erysipelas plays an important part in the etiology. The latter assumption has not been borne out, though in a few cases the onset has been coincident with erysipelatous attacks, for instance, two of the cases of hyperostosis in epileptics related by Sommer (84). Virchow called attention to the fact that the hyperostotic bone was sometimes described as soft and spongy, but more commonly as hard and ivory-like, and showed that the latter condition is of two kinds, either an osteosclerosis which is secondary to the spongy condition or an eburnation which is primary. A concentric arrangement of the lamellæ indicates an osteosclerosis, one parallel to the surface eburnation.

The case of Bickersteth, fully reported by Murchison (70), is probably to be considered one of leontiasis, although the hyoid bone and the fibula were also involved. At the age of fourteen years enlargement of the facial bones commenced, that of the fibula at twenty-seven. Death occurred at thirty-four, preceded by symptoms of compression of cranial contents, severe headaches, exophthalmos and blindness of the left eye. At the autopsy the superior maxillary bones were found enlarged, the inferior maxillæ enormously so. All bones of the cranial vault were involved except the occipital. The skull was covered externally with excrescences, the orbits, nasal and external auditory openings reduced in size. Histologic examination was confined to the fibula, "as it was not considered necessary to injure the cranium."

Wrany (93) reports a case where the disease commenced in the lower jaw, which is unusual. The skull was thickened and hard, the sutures obliterated internally. The frontal bone measured 12 mm., the parietal 6 mm., the occipital at the inion 20 mm. in thickness. The posterior wall of the sella turcica was atrophic and perforated. The inferior maxilla was thickened in all parts except the condyles, the superior maxillæ and lachrymal bones were also involved. No bone outside the skull was involved.

Le Dentu (56) reports a case of hyperostosis of the facial bones of two years' duration in an eighteen-year-old girl. A piece was excised and pronounced to be sarcomatous by competent men at the College de France. Le Dentu did not accept the diagnosis as healing took place after excision, and designates the condition a "diffuse non-syphilitic periostosis." However, Baumgarten and Schmidt are inclined to consider the diagnosis of sarcoma correct. This controversy is of interest in connection with our own case, where we evidently have to deal both with leontiasis ossea and sarcoma.

Skull No. 205 in the Museum of the Breslau Pathologic Institute, described by Fischer (59) as an instance of leontiasis, is not mentioned in the treatises of Baumgarten (53) and Schmidt (82). The left side of the frontal and both parietal bones were involved. The tumor-like thickening of the frontal bone measured 36.5 cm. in circumference, 15 cm. in length, 6.5 cm. in height, 12.5 cm. in width. The bone was of ivory hardness, uneven, the inner surface covered with coarsely granular osteophytes. The cavity of the skull was very little narrowed. He also mentions the case of a man, eighteen years old, with a similar tumor the size of a fist at the right frontal eminence, which had existed for six years without any symptoms of compression. Fischer also describes the skull of a woman, who died of pulmonary tuberculosis, with bony tumors of the left temporal bone, sphenoid bone, and left side of the lower jaw.

In 1881, Paquet (72) presented before a surgical society a woman, twenty-two years old, on whose left upper jaw a tumor was first noticed at the age of three years. It had grown rapidly the last two years only, before that very slowly. The left eye was compressed and nasal fossa almost obliterated. The tumor was resected and histological examination showed most of it to be hyperostosis, but it was sarcomatous in the region of the antrum and palate. Baumgarten is of the opinion that two distinct affections were present, namely a hyperostosis, probably congenital, and a sarcoma of two years' standing.

The osteoporotic and much thickened parietal bone found in an Egyptian tomb, preserved in the Museum of the Royal College of Surgeons, London, and described by Eve (58), if an instance of leontiasis, is probably the oldest specimen of that disease in existence.

Silcock (83) reported the case of a man, who, at the age of twenty-three received a blow on the left forehead followed in five days by epileptic attacks and in a year by a bony tumor. A year later, when it had reached the size of a Tangerine orange, trephining was performed, the bone found 35 mm. thick and beset with small exostoses, and the left frontal sinus obliterated. No histologic examination was made, a fact to be regretted, as it would be desirable to establish histologic points of differentiation, if possible, between such rapidly growing exostoses and the slowly advancing typical leontiasis.

In the same year (1890) appeared Poisson's (74) account of a patient with onset at sixteen of hyperostoses of the superior maxillæ and obliteration of nasal cavity and antrum. Histologic examination showed a rarefied condition of the bone. A trophic disturbance of neuropathic origin is considered as the basis of the trouble.

In 1892 F. Baumgarten published his thesis entitled, "La leontiasis ossea" (53), in which a thorough review of nearly all recorded cases is given, together with a discussion of the etiology, pathology, symptoms, and diagnosis of the affection. No new cases are reported, but further details given of a case previously reported by Pean (73), who had extirpated multiple tumors of the palate, upper and lower jaws in a woman, twenty-two years old. At the age of nine the first tumor had appeared in the right palatal vault. Histologically all the tumors presented the same structure. Baumgarten hesitates whether to call them fibrosarcoma, osteo-fibroma, or osteo-fibro-sarcoma. The tumor of the lower jaw was surrounded by a fibrous capsule. There was no extension to the cranium. Baumgarten does not consider this a case of leontiasis. However, considering the long duration, it seems to the writer that this may be another instance of sarcoma arising in hyperostotic bone.

Starr (86) reported a case of leontiasis ossea or, as he proposed to call it, "megalo-cephalie," in a woman fifty-two years old, with onset at the unusually late age of forty-four. There was general hypertrophy of the bone over the entire calvarium, more marked just behind the temples. The head measured 24 inches in circumference. The cavities of the nose and mouth were encroached upon, the soft parts of the head and neck also thickened. Many nervous symptoms were present. Thyroid extract was tried without effect.

In 1895, Horsley (63), reported five cases of leontiasis ossea which he defined as "the development of bony new formation resembling an overgrowth of diploë, and attacking the cranial bones (by preference the frontal), or more rarely the facial bones, especially the jaws." In a summary he says: "In all five cases the disease began in childhood or in early youth. In the three cases in which the disease attacked the eyebrow and roof of the orbit early, the displacement of the eyeball downwards occurred before any notable swelling appeared above in the supraorbital ridge. In the same three cases the region of the external frontal process was the focus of the malady. In no case was there any evidence of syphilis nor any really direct history of traumatism. In all cases the prominent symptom of distress was the pain produced by the entanglement and pressure upon the branches of the fifth nerve passing through, or by the affected bones. In the cases where operation was possible the mass was readily removed piecemeal, starting from the border of conjunction with the healthy bone."

Case 1. Male, age 19, onset at 15 with swelling above left brow, headache, vomiting, epileptic fits. Thyroid gland enlarged. Swelling of vertex, affecting frontal, right temporal, part of parietal and occipital bones. No operation. Case 2. Female, age 26, for five years swelling of left side of head. In 1888 the thickened bone was removed by operation. Recurrence, a second operation in 1894, with relief of all symptoms. Case 3. Male, age 28, onset at 20 with exostoses on each side of nose. Five years later protrusion of right eyeball and optic atrophy. Right superior and inferior maxillary bones were resected. Three years later operation on left side of face which had become involved. Case 4. Female, age 13, swelling of left side of frontal bone, resected. Case 5. Male, age 35, swelling of right side of frontal bone for at least 17 years. Resection of diseased portion of bone with relief of pain. Horsley gives the following anatomic description: "Outer surface of tumor in every case smooth, periosteum thickened. When the latter was stripped off the diseased bone presented a marked contrast to the normal, in being notably more vascular, the surface being extensively filled with minute foramina, the branching of superficial vessels very prominent, and the color a deep red as compared with that of normal bone. There was no demarkation between the tables and the diploë. Histologic examination was complete only in cases 2 and 5. The outer, rather harder portion of the bony mass consisted of normal trabeculæ of bone separated by unusually wide medullary spaces. The lamellæ in the bone trabeculæ were well marked, but homogeneous throughout from the margins of the medullary spaces inward. In this region the medullary spaces were filled with a delicate, almost myxomatous connective tissue in the meshes of which hardly any characteristic medullary corpuscles appeared, except at the periphery of each space, where a complete row of osteoblasts was present in most cases. The central, softer and more friable portions of the tumor presented very different appearances from the foregoing. The medullary spaces were very large, the bony trabeculæ in process of active absorption and medullary tissue replaced by a neoplastic, fibrous growth. To consider these changes in further detail the alteration in the marrow may be taken most conveniently first in order. The medullary tissue of the outer portions of the tumor has already been stated to consist chiefly of a delicate fibrous tissue. From this condition to one in which the whole medulla was converted into a dense feltwork of fibrous reticular substance, every stage could be found. In the progress of this metamorphosis the individual fibers appeared to become thickened and to take a brownish stain with hematoxylin. While in the extreme stages the greatly thickened fibers assumed a more granular appearance, the whole suggested an osteogenic condition. Soon after the fibers became denser the osteoblasts disappeared and their place was taken by osteoclasts. The bony trabeculæ, as soon as the medullary tissue became obviously altered, showed notable changes, until in the most advanced areas of disease every bone trabecula exhibited by change in the staining reaction (deeper color) and morphological alteration of the cells, a marked reversion to a chondroid stage, and furthermore the margins and the surfaces of the trabeculæ displayed many false Howship's lacunæ." The extreme rarity of histologic descriptions renders the reproduction of this one in extenso justifiable. Horsley concludes by saying: "Leontiasis ossea is a pathologic entity in which both osteoplastic and osteoclastic processes are represented, and that it is a hopeless condition, for which operation, if extensively practiced, offers considerable relief."

In 1896 a valuable article, entitled "Hyperostosis cranii," by Putnam (76), appeared in this country, with four new cases, statistics of fourteen typical cases, and a brief critical review of the literature. Case 1. Female. Mother and sister had "suffered with their heads." At three, some disease of ankle, followed by separation of pieces of bone. Bright in youth but inclined to drowsiness. Onset at 19 with headache, tinnitus, later deafness, head gradually broadening. For years a purulent discharge from ears. Bilateral facial paralysis before death, also dimness of vision in one eye. Died at about 30. Autopsy showed a general purulent periostitis cranii. Diffuse thickening of cranium, most marked anteriorly, 1.5 cm. Bones with a worm-eaten appearance from caries. Weight of skull nearly twice the normal. Orbits and sella turcica smaller than normal. Grooves for vessels practically absent. Diploë ivory-like. Case 2. Male. Two bean-sized lumps noticed at the top of the head at five. They finally reached the size of 2½ inches in diameter and ¼ inch in height. Prominent occipital protuberance and temporal ridges. Slight optic neuritis, eyes prominent. Epileptiform convulsions began at seven; deafness and dementia before death, at 22. No autopsy. Case 3. Man, observed at 50 years, had noticed that his head was growing larger for ten or twelve years, enlargement attended by neuralgic pains, impaired hearing, insomnia. Case 4. Female, age 35. Enlargement of head first noted in the region of left parietal eminence at 23. Burning sensations and numbness of face. Circumference of head 65.5 cm. Surface of cranial bones irregular from presence of exostoses. A case observed by Dr. Prince is also reported by Putnam in the same article. Male, struck on the forehead by a pulley, two years after noticed swelling of frontal bone, followed by symptoms of compression and death six years after the injury. The autopsy showed marked thickening of the skull in the mid-frontal region, of the temporal ridges, and particularly of the orbital plates.

Following Putnam's article is a report by Edes (57) of a woman, age 52, whose head had been growing larger for some years. Six years later (1902) this case was found by Prince (75) to have developed into one of ostitis deformans, almost all of the long bones in the body having become affected. There was also a general myasthenia.

White's case (92) was that of a man, 32 years old, with a prominent bony ridge running horizontally round the back of the head, reaching within an inch of outer canthi of both eyes. This had appeared a short time after a fall from a window at the age of three years. Later in childhood he had fractured the left femur, at 22 the right femur, at 29 the right shoulder became stiff. The maximum circumference of the head was 24½ inches. Facial bones normal. Slight nystagmus and bilateral deafness; no headache. General health good. According to Prince (loc. cit., p. 810), this case later came to autopsy, recorded by Collier. Unexpectedly a condition of syringomyelia was found which had not given rise to the usual sensory symptoms. Prince thinks the bony changes were very likely of the nature of the trophic lesions dependent on that disease.

Noble (71) reported a case of a young man with extreme bulging of the nose and lower part of the forehead, the eyes being five inches apart. The disease had commenced with the appearance of a bony growth in the upper part of the right nostril, four months after the injury of the nose by a fall at the age of eighteen.

Young's patient (94), a man who died at 46, had prominent superior maxillary bones and supraorbital ridges with obliteration of the antra and frontal sinuses, and narrowing of the right orbit and both nasal cavities. Cranial bones of normal thickness, sella turcica normal.

Hinsdale's (62) patient, a woman, died at 71. There was diffuse hyperostosis of the skull, thickness varying from 14 mm. in temporal, to 35 mm. in occipital bone; circumference of head 71 cm. Diploë reduced in amount, bone unusually soft and friable. The skull-cap weighed 1870 grams. The lower half of the head was not enlarged. The epiphyses of both femurs were enlarged and covered with friable exostoses, and the long bones of the lower limbs were slightly bowed. Acromegaly or myxedema were excluded. The enlargement of the head was first noticed at sixty-four.

Sommer (84) contributes an article on cranial hyperostoses in the insane. Epileptics are particularly frequently affected. The anterior half of the base, especially the sphenoid, is most frequently involved. Twenty cases were studied.

Stack's case (85) was that of a woman who, at the age of three years, injured the forehead by a fall. The wound suppurated. Enlargement of the head was first noticed at the age of seven. She died at 21. At the autopsy the skull was found greatly thickened, the half skull without the lower jaw weighing 7½ lbs. The thickening was greatest in the frontal bone. The bone was very soft. The lower jaw weighed 1 lb., and is described as follows: "The whole of the horizontal ramus was enlarged and rounded; the compact shell was expanded and filled with very porous bone, containing a quantity of very slimy, gummy material, which poured out when cut into." The nasopharynx contained a firm white tumor attached to the hard and soft palate, found on histologic examination to be a fibro-sarcoma with numerous myeloid cells. Brain and hypophysis normal. There were scoliosis, a soft and distorted pelvis, bent femora and tibiæ, changes which led Prince to suspect that the case was a combination of hyperostosis and osteomalacia.

Schiller (80) reports a case from Czerny's clinic. Male, age 30. A brother was delivered by embryotomy on account of abnormal size of thorax. Patient delivered by forceps as head was unduly large. Abnormal increase in size of head continued until age of 19 years, when it became stationary. The only symptom was frequent headache. The circumference of the head was 61 cm. The head was weighed, hanging over the edge of the table, when the patient was anesthetized. Its weight was between 5 and 5.5 kilograms, while that of normal heads in the same position was 2 to 2.5 kilograms. There were prominences of the frontal and parietal bones, seen in X-ray pictures to be due to actual bony thickening. Facial and other bones not involved.

In the appended table a summary of the causes just recorded is given. Although they undoubtedly do not constitute a clinical entity, in our present state of knowledge it seems convenient to consider them together as all more or less possess the characteristics of Virchow's leontiasis ossea. In many of them, such as some of Horsley's cases, there may be only a resemblance to leontiasis, in others the latter may be a part of a more general disease, but as the nature of leontiasis itself, as well as of nearly all of the allied disorders, is unknown, only arbitrary distinctions can be made, when the phenomena overlap. Hence, we will consider these cases together, first as to their

*Etiology*—Heredity seems to play no part. The sexes are about equally affected. Onset nearly always in childhood or early youth, even before birth, if we include Breschet's and Schiller's cases. Of the five cases with onset after twenty-five, those of Starr, Edes, and Hinsdale were complicated by disease of other bones, and possibly belong to ostitis deformans. Trauma is said to have preceded the onset six times. Of these cases Buhl's was complicated by gigantism, Stack's by osteomalacia, White's probably was due to syringomyelia, Noble's and Silcock's were of such rapid course as to suggest a true tumor. The erysipelas and suppurative inflammations present in a few cases were evidently merely complicating or accidental. Baumgarten's view that the disorder is trophic and developmental is probably the best at present.

*Morbid Anatomy and Histology*—Sternberg (87) divides the cases into ***diffuse hyperostoses*** with involvement, rarely even, of all cranial and facial bones (cases of Malpighi, Jadelot, Sandifort, Ilg-Gruber, Gaddi), and localized ***tumor-like hyperostoses,*** such as the case Forcade, and those of Buhl and Bickersteth-Murchison. Such a line, however, seems rather artificial. As to the bones involved, the following may be learned from 30 fairly completely reported cases in our table:

| | |
|---|---|
| Cranial, upper facial and lower jaw | 10 |
| Cranial only (or at least principally) | 10 |
| Cranial and upper facial | 4 |
| Cranial and lower jaw | 2 |
| Upper and lower jaws | 3 |
| Upper jaw only | 1 |

Of the cranial bones the anterior part of the frontal is usually most affected. Of 22 cases the disease is stated to have commenced in the cranial bones 11 times, upper facial 10 times, and lower jaw once (Wrany's case). The affected cranial bones are generally harder than normal, with absence of diploë, and obliterated sutures. Exceptions are the cases of Stack, Hinsdale, Buhl, and our own, where the hyperostotic cranial bones were abnormally soft. The weight of the skull is often enormously increased, as well as the thickness of the vault, as the frontal bone in our case, 85 mm. The external surface is generally rough, often with large exostoses, while the internal surface is most commonly smooth. Narrowing of the cranial cavity, orbits, nasal cavities, sinuses, and foramina for nerves and vessels is common. The affected facial bones are usually the seat of nodular exostoses, diffuse thickening being much rarer than in the skull. Histologic examinations are too few to admit of any generalizations. The association with sarcoma in as many as five cases, those of Paquet, Stack, Pean-Baumgarten, Le Dentu, and ours, must be more than a coincidence. The descriptions in these cases lead us to infer that the sarcomatous process started in the periosteum of the diseased bone. In only one of these cases, that of Le Dentu, is it possible that the entire process in the bone was due to sarcoma. One may also suspect that some of the more acute cases following injuries, as those of Silcock and Noble, really were instances of osteosarcoma. The complications with gigantism (cases of Buhl, Sirena (42) and ours), with syringomyelia (White), osteomalacia (Stack), ostitis deformans (Edes), and acromegaly (Saucerrotte), and possibly a case of Sternberg's (87), are extremely interesting and suggest an etiologic relationship between all these obscure conditions.

*Symptoms*—Most of these are explained by pressure on the brain or nerves. Headache is the rule. Anosmia, blindness, ocular paralysis, neuralgic pains, numb or anesthetic areas, tinnitus, deafness, dysphagia, speech troubles, epileptiform convulsions, mental disorders, have all been recorded. Optic neuritis was recorded in Putnam's case, that of Edes, and ours. The course is very slow, but nearly always progressive, usually extending over ten to thirty years.

*Diagnosis*—*Ostitis deformans* usually begins after forty, and the long bones are here involved. When it begins in the cranium, a differential diagnosis is extremely difficult or impossible until the long bones become involved. Recently Prince (75) has advocated the view that the two perhaps are only different manifestations of the same disease, and probably trophic disorders, that the underlying cause may be found in the nervous system which he makes a plea to examine more closely in the future. *Acromegaly.*—The enlargement of the hands, feet, tongue, and other soft parts, makes a distinction easy in a pronounced case, but, as stated, here we also have combinations of the two. *Sarcoma.*—Diagnosis may be very difficult when the maxillary bones are affected, particularly in the case of multiple, slowly growing tumors. Slow growth and gradual enlargement of the skull are in favor of hyperostosis. The tendency for sarcoma to arise from hyperostotic bone must be borne in mind.

*Prognosis*—Unfavorable, as the tendency is for the encroachment on cavities and foramina to progress steadily.

*Treatment*—Very little is to be expected until the underlying morbid tendency can be directly antagonized. Operation may afford relief or even cure, if performed early, but in most cases the disease is too diffuse for surgical interference, and the reappearance of the disease at some other point is likely when excision of the primary focus may be possible. This indeed is to be expected if we are dealing with a developmental trophic disorder.

In conclusion the writer wishes to thank Professor Hektoen for placing the autopsy material at his disposal and for much valuable aid and advice.

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### LEONTIASIS OSSEA, ETC.

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| OBSERVER | SEX | AGE AT ONSET | AGE AT DEATH | BONES OF SKULL AFFECTED | OTHER BONES AFFECTED | PART FIRST AFFECTED | CONDITION OF DISEASED BONE | CONDITION OF CRANIAL SUTURES | SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES | REMARKS |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. Malpighi | ? | ? | ? | All cranial particularly temporal, tumor of cherry size near sagittal suture. Orbits small. | ? | ? | ? | Effaced. | ? | Skull weighed 3,618 grams. Nothing else observed. |
| 2. Jourdain, Ribel (Case Forcade). | M | 12 yrs. | 45 yrs. | Upper and lower jaws, nasal, malar, all cranial bones. Orbits small. | All unusually delicate. | Right upper jaw. | Covered with large, hard exostoses. | — | Blindness. Speech troubles. | Onset with abscess at inner canthus. Dried skull weighed 4,192 grams. Lower jaw alone 2,792 g. Facial muscles fibrous and adherent to periosteum. |
| 3. Sandifort. | ? | ? | ? | All cranial. | ? | ? | Skull thickened by one inch. Abundant diploe. Exostoses on inner surface, particularly of frontal bone. | — | ? | Skull only observed. |

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|---|---|---|---|---|---|---|---|---|---|---|
| 4. Jadelot. | ? | ? | ? | All cranial. | ? | ? | Thickness 19.8 to 40.2 mm. Very compact. | — | ? | Skull weighed 3,913 grams. |
| 5. Saucerrotte. | M | ? | 39 yrs. | Cranial and lower jaw. | Spine, ribs, sternum, clavicles. Bones of extremities. | ? | | — | ? | Lips were thick. Circumference of head 797 mm. Probably acromegalic. |
| 6. Ilg-Gruber. | F | 10 yrs. | 27 yrs. | All cranial, most so occipital. Sella turcica shallow. All foramina small. | ? | ? | Thickness of cranial vault 19.8-52.6 mm. Bones hard with exostoses. | Mostly disappeared. | Blindness, deafness, dysphagia, anosmia, headache, delirium, epileptic fits. | Skull without lower jaw, teeth and nasal bones weighed 4,200 g. |
| 7. Kilian. | ? | | | All cranial. | | | Thickness 27-54.1 mm., latter parietal. | — | | |
| 8. Bojanus. | | | | | | | Compact. No diploe. Thickness 27-67 mm. | Mostly disappeared. | | Skull without lower jaw weighed 4,000 g. |
| 9. Breschet. | | Before birth. | 18 mos | All cranial, facial not involved. | Probably none. | ? | Greatest thickness of vault 1 inch. Ant. fontanelle present. | ? | Convulsions since birth. | |
| 10. Vrolick. | ? | ? | ? | All cranial. Sella turcica narrow and deep. | ? | ? | Thickness 7-20 mm. | Obliterated except squamous. | ? | Skull weighed 1,305 grams. |
| 11. Schützemberger | | ? | 40 yrs. | All cranial. Foramina small. | | | Hard, thickened at expense of inner table. No diploe. | Obliterated. | Convulsions, headache. Loss of speech. Mental enfeeblement. | |

| OBSERVER | SEX | AGE AT ONSET | AGE AT DEATH | BONES OF SKULL AFFECTED | OTHER BONES AFFECTED | PART FIRST AFFECTED | CONDITION OF DISEASED BONE | CONDITION OF CRANIAL SUTURES | SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES | REMARKS |
|---|---|---|---|---|---|---|---|---|---|---|
| 12. Huschke. | F | ? | 17 yrs. | All cranial. | ? | ? | Thickness 1-4 cm. latter occipital. Hard, little diploe. |  |  | Skull weighed 3,224 grams. |
| 13. Gaddi. | ? | ? | ? | Facial and cranial. | Axis enlarged. | — | Surface rough. | Obliterated. | ? | Skull weighed 3,660 g., lower jaw 422g. Skull and axis only bones examined. |
| 14. Bickersteth. Murchison. | M | 14 yrs. | 34 yrs. | Cranial except occipital. Upper and lower jaws. | Hyoid and fibula. | Facial bones. | Skull covered with excrescences. |  | Headache, blindness |  |
| 15. Wrany. |  |  |  | Cranial and facial. | None. | Lower jaw. | Hard. Thickness of cranial 6-20 mm. | Obliterated internally. | Insanity. | Preceded by trauma. |
| 16. Buhl. | M | 9 yrs. | 25 yrs. | Facial and cranial. Left orbit small. | Scoliosis. | Facial bones? | Frontal 6 cm. thick, soft. Many exostoses on facial bones. |  | Death ascribed to cerebral compression. | Gigantism, hgt. 235 cm. Onset after kick on cheek. |
| 17. LeDentu | F | 16 yrs. | ? | Facial. | None. | Facial bones. |  | ? | None. | Operation. Probably sarcomatous. Observed at 18. |

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|---|---|---|---|---|---|---|---|---|---|
| 18. Fischer. | ? | ? | ? | Frontal and parietal. | ? | ? | Uneven, hard. Large exostosis of frontal bone. | ? | ? |  |
| 19. Fischer. | M | 12 yrs. | ? | Frontal bone. | None. | Frontal bone. | Tumor size of fist at right frontal eminence. | ? |  | Observed at age of 18. |
| 20. Paquet. | F | 3 yrs. | ? | Left upper jaw. | Probably none. | Left upper jaw. | Histologically partly hyperostosis, partly sarcoma. | ? |  | Observed at 22. Growth rapid last two years. Operation. |
| 21. Silcock. | M | 24 yrs. | ? | Left frontal. | None. | Left frontal. | Tumor of frontal 35 mm. thick. Small exostoses on inner surface. Frontal sinus obliterated. | ? | Epileptic fits. Commenced 5 days after injury. | Observed at 25. Onset one year after blow on head. Operation. |
| 22. Poisson. |  | 16 yrs. |  | Upper jaw. | Probably none. | Upper jaw. | Hyperostosis of superior maxillary bone. Obliteration of nasal cavity and max. sinus. | ? |  | Histologic exam. showed a rarefying ostitis. |
| 23. Pean, F. Baumgarten. | F | 9 yrs. | ? | Hard palate, upper and lower jaw. | None. | Hard palate. | Multiple tumors. | ? | None. | Histologically, between fibrosarcoma, osteofibroma and osteofibrosarcoma. Observed and operated at 22. |

| OBSERVER | SEX | AGE AT ONSET | AGE AT DEATH | BONES OF SKULL AFFECTED | OTHER BONES AFFECTED | PART FIRST AFFECTED | CONDITION OF DISEASED BONE | CONDITION OF CRANIAL SUTURES | SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES | REMARKS. |
|---|---|---|---|---|---|---|---|---|---|---|
| 24. Starr. | F | 44 yrs. | ? | Calvarium and facial bones. Nasal and oral cavities smaller. | Cervical vertebrae |  |  |  | Nervousness, irritability, numbness of fingers, uncertain movements. | Observed at 58. Circumference of head 24 in. Skin thickened. |
| 25. Horsley. | M | 15 yrs. | ? | Entire vertex. | None. | Frontal bone left side. | Smooth externally with minute foramina. More vascular than surrounding bone, no demarkation between tables and diploe. Periosteum thickened. | ? | Epileptic fits, headache and vomiting. | Observed at 19. |
| 26. Horsley. | F | 21 yrs. | ? | Left side of head |  | Frontal bone left side. |  |  | Pain. | Operation twice. Observed at 26. |
| 27. Horsley. | M | 20 yrs. | ? | Nasal, superior and inferior max. bones. |  | Nasal bones. |  |  | Pain. | Operation at 23. Recurrence; 2d operation at 28. |
| 28. Horsley. | F |  | ? | Frontal bone. |  | Frontal bone left side. |  |  | Pain. | Operation at 13. |
| 29. Horsley. | M | 18 yrs. | ? | Frontal bone. |  | Frontal bone right side. |  |  | Pain. | Operation at 35. |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| 30. Putnam. | F | 19 yrs. | 28 yrs. | Cranial and malar bones. Orbits smaller. Sella turcica narrow. | None. | Cranium. | Diffuse thickening of cranium, most marked ant. Extensive caries. Diploe ivory-like. | Obliterated. | Headache, pain in ears and eyes. Deafness. Facial paralysis. | Suppurative periostitis cranii. Weight of skull nearly twice the normal. |
| 31. Putnam. | M | 5 yrs. | 22 yrs. | Frontaland parietal. | None. | Frontal and parietal. | Two broad exostoses on top of head. Occipital protuberance and temporal ridge prominent. | ? | Epileptic fits. Deafness. Optic neuritis. Dementia. | No autopsy. |
| 32. Putnam. | M | 38 yrs. | ? | Cranial bones. Edges of orbits thickened Facial bones uninvolved. | None. | Cranial bones. | | ? | Neuralgic pains. Throbbing sensation in rt. ear. Impaired hearing. | Abscess of both ears at 35. Observed at 50. |
| 33. Putnam. | F | 23 yrs. | | Cranial bones. | None. | Left parietal bone. | Calvarium covered with exostoses. | ? | Burning sensation in head, numbness of face. Sometimes diplopia. | Observed at 35. Circumference of head 65.5 cm. |
| 34. Prince. Putnam. | M | Adult. | | Frontal bone. | | Frontal bone. | Frontal bone thick, particularly orbital plates. Temporal ridge prominent anteriorly. | ? | Frontal headache,drowsiness Mental dulness. Vomiting. Rt. optic atrophy. | Onset two yrs. after injury to frontal bone. Death 4 yrs. after onset. |
| 35. Edes. Prince. | F | 40-50 yrs. | | Cranial bones, mainly frontal. | Nearly all long bones. | Cranial bones. | Spine and long bones deformed. | ? | Weakness of gait. Deafness. Rt. eye nearly blind. | First observed at 52. Probably ostitis deformans. |

| OBSERVER | SEX | AGE AT ONSET | AGE AT DEATH | BONES OF SKULL AFFECTED | OTHER BONES AFFECTED | PART FIRST AFFECTED | CONDITION OF DISEASED BONE | CONDITION OF CRANIAL SUTURES | SYMPTOMS OF COMPRESSION OF BRAIN OR NERVES | REMARKS |
|---|---|---|---|---|---|---|---|---|---|---|
| 36. White. Collier. Prince. | M | 3 yrs. | Abt 35 yrs. | Cranial only. | None. | Cranial bones. | Projecting ridge around back of skull. | ? | Bilateral deafness. Slight nystagmus. Fits. Spastic atrophic paralysis of arm. | Onset following injury. Operation for fits. Autopsy showed syringomyelia. |
| 37. Noble. | M | 18 yrs. | 21 yrs. | Nasal, frontal, sup. max. bones. | None. | Right nasal bone | Extreme bulging of lower part of head. | ? |  | Onset 4 mos. after fall on nose. |
| 38. Young. | M | 39 yrs. | 46 yrs. | Sup. max. and frontal bones. Rt. orbit and both nasal cavities diminished. | None. | Left sup. maxillary. | Symmetrical prominent bosses of sup. max. and frontal bones. Vault of normal thickness. Frontal sinuses and antra occluded. | ? | Three attacks of insanity. Anosmia. Dimness of vision. | Bosses of sup. max. bone found cancellous on section. Marked atheroma of aorta. |
| 39. Hinsdale | F | 64 yrs. | 71 yrs. | All cranial. | Slight bowing of the long bones of extrem. Exostoses of femurs. | ? | Extremely soft. Diffuse hyperostoses of cranium. Thickness of vault 14-35 mm. |  |  | Skull cap weighed 1,870 gms., brain 1,360 gms. Circumference of head 71 cm. |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| 40. Stack. | F | 7 yrs. | 21 yrs. | All cranial.<br>Lower jaw.<br>Palate. | Scoliosis. Soft and distorted pelvis, femurs and tibiæ bent and soft. | Cranium. | Bones soft. Fibro-sarcoma of naso-pharnyx. | | | The half skull without lower jaw weighed 7½ lbs., lower jaw 1 lb. Injured forehead at 3. Probably complicated by osteomalacia. |
| 41. Schiller. | M | Before birth. ? | | Frontal, parietal and occipital bones. | None. | ? | Tumor - like prominences of frontal and parietal bones. Prominent ext. occipit. prot. | Not involved so far as palpated. | Headache. | Observed at 30. Circumference of head 61 cm. Birth difficult on account of size of head. |
| 42. Present Case. | M | 9 yrs. | 28 yrs. | All cranial.<br>Left facial. | Scoliosis. | L. frontal. | Cranial bones soft, thick, invaded by sarcoma. Frontal 85 mm. thick. | Prominent. | Headache, blindness, immobility of eyeballs. Ptosis. Facial anesthesia. Deafness, tinnitus. | Gigantism, hgt. 245 cm. |

# STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL HUMAN BRAIN AND IN DEMENTIA PARALYTICA.

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*(Continued from page* 554.)

*Technique.* Turning to the technique employed in this work, first will be described a method used in transporting material considerable distances in the minimum amount of space, and preparing the same in the shortest possible time.

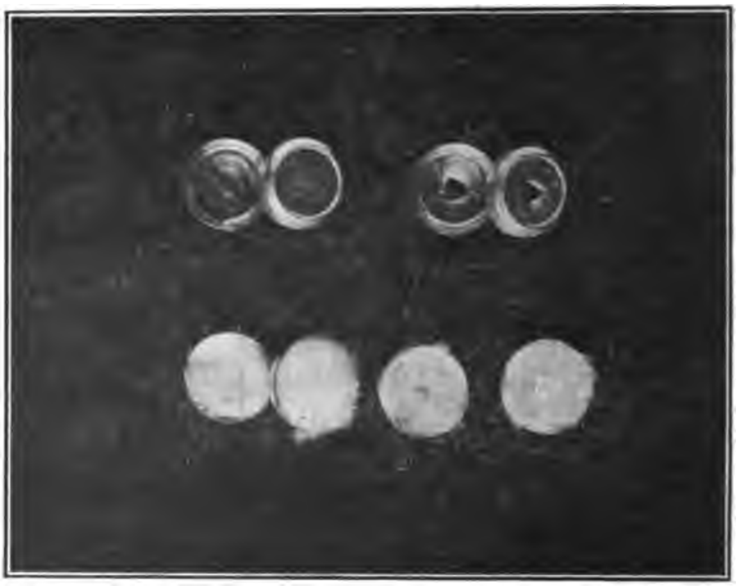


Fig. A.

On several occasions the writer was enabled to secure more than one brain at the same time, and at a considerable distance from the laboratory. It was desired to place small blocks from various parts of each of the brains in various fixative media in the shortest possible time, and to put them into the smallest possible space for transportation. The smallest size of tin boxes known as "Miller's patent seamless box" (Fig. A) were secured at trifling cost at Eimer & Amend's, in New York city; though any small box would answer the purpose. These measured but 2.5 c. c. in diameter and 1.25 c. c. in depth. One gross of these boxes occupy only a space of 15 c. c. square by 5 c. c. in depth. By piercing a hole in both the cover and bottom of these boxes a free circulation of the fixative medium is secured—as mentioned, any other small box or phial admitting of the free circulation of the fixative medium could be used; but these chanced to be the most convenient and available receptacles to the writer. By placing a bit of absorbent cotton or cheesecloth in both the bottom of the box and in the cover the specimen is perfectly protected. A square box of black pins (Fig. B), such as can be secured at any drygoods store at trifling cost, was purchased. Small squares of cardboard

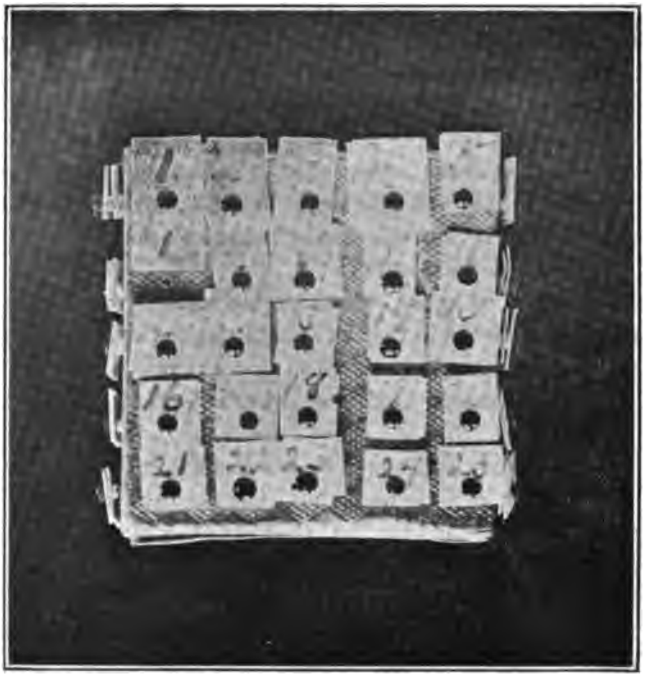


Fig. B.

were cut out and numbered in duplicate, using a lead pencil. Each number, with its single duplicate, was placed upon a pin and returned to its position in the box. The various fixative fluids desired were carried in small glass jars with ground glass covers. All these preparations are made in advance so that at the autopsy, upon removing the brain, small pieces can be taken from any part of the cortex, each one placed in a separate small tin box with a number from one of the pins. The other or duplicate number is left upon the pin, and the latter thrust into the space on the brain surface from which the block was taken (Plate I, Fig. 1). The small tin box is then thrown into whatever fixative fluid is desired, and the fluid enters and fills up the interior by means of the openings above described, the whole process taking less time than required in explanation. In this way a large number of blocks can be taken from one or more brains without any possibility of confusion, and in a minimum amount of time, and likewise take up a minimum amount of space, requiring no writing or labeling of specimens at the time. After all blocks desired are removed the brain is carefully placed upon cotton in a tin pail of the required size in whatever fixative fluid is desired, and other brains can be treated similarly.

At the laboratory subsequently the exact locality from which blocks were taken can be noted at leisure in the most accurate manner. The writer has by this method taken blocks from various parts of two separate brains, placed them in half a dozen different fixative fluids, and packed them up, together with the two brains in tin pails of suitable diameter, placing everything in a hand bag of medium size, and carried the same many miles on the train without the slightest inconvenience or knowledge by others of the contents of the hand-bag. The fixative agents employed in this work were alcohol absolute, alcohol 95 per cent, formalin 10 per cent solution (40 per cent formaldehyde, 1 part by volume, water 9 parts by volume), Van Gehuchten's fluid (alcohol absolute 60, chloroform 30, and glacial acetic acid 10 parts by volume), and Lang's solution (mercuric chloride 5 grams, sodium chloride 6 grams, acetic acid 5 grams, and water 100 grams). The most satisfactory results have come from fixation in Van Gehuchten's fluid, and the alcohols. In the employment of the latter more or less shrinkage results, but this can be recognized, and the chromatic substance within the cell is usually well marked. Van Gehuchten's fluid was used as follows: Small blocks, not more than .3 to .5 c. c. in thickness, were immersed in Van Gehuchten's fluid and left for twelve hours. They were then placed in 95 per cent alcohol, where they remained until desired for use. No changes in the contour of the cells were observed to result, and the chromatic substance in these cells was well shown in the subsequent staining. Blocks to be imbedded were then placed in absolute alcohol, and, for the paraffin method, transferred to xylol, and left in the latter for several hours till thoroughly permeated. They were then placed in paraffin—melting point about 50° C., for from 45 minutes to one hour, and then transferred to another paraffin bath of the same melting point for the same length of time, so as to secure complete penetration and the removal of all xylol. The blocks were then imbedded and subsequently sectioned by a Minot microtome serially. The sections were made varying in thickness from 2 to 15 microns, those from 6 to 10 microns being found best for study of the arrangement and internal structure of the cells. The celloidin method was also employed, but it was more difficult to secure thin sections when desired, and especially to arrange them serially. A thin smear of egg albumin was placed upon a perfectly clean slide, several consecutive sections placed upon the same, and then a small quantity of water by means of a pipette allowed to flow under the sections. This was gently warmed upon the water bath or over an alcohol lamp until the sections were perfectly flattened out. The water was drawn off and the sections allowed to dry. This treatment permitted all subsequent manipulations with the sections upon the slides, without their floating off or becoming disturbed or injured. The slides were then placed in xylol to dissolve out all paraffin and run down in successive grades of alcohol from absolute alcohol to 30 per cent alcohol, and from the latter immersed in water. The sections were then stained upon the slide by means of an aqueous solution of methylene blue, as given by Nissl (methylene blue, 3.75 grams; Venetian soap, 1.75 grams, and distilled water 1,000 c. c.), or by a 1 per cent aqueous solution of methylene violet or a 1 per cent aqueous solution of thionin. A counter stain of erythrosin was used in some cases after treatment with methylene blue, giving the achromatic substance a pink color in contrast to the blue color of the chromatic substance. The routine method was to gently heat the slide covered with the stain for two minutes over an alcohol lamp, keeping the slide in constant motion, and only allowing it to become sufficiently heated so that the steam would come from the surface, but no bubbling of the dye in solution. The dye was then gently washed off, the slide immersed in water, and then run up into 30, 50, 70, 80, 95 per cent. alcohol, absolute alcohol plus xylol equal parts, xylol, and finally mounted in xylol-damar. Some slides were placed, after immersion in water following the staining process, in anilin-oil 10 parts, absolute alcohol 90 parts, and subsequently treated as described by Nissl, and finally mounted in benzene-colophonium. The first method, however, gave the most satisfactory results in the hands of the writer, and which, as seen, embodies various modifications of the original Nissl method. Sections prepared in the routine method above described have been frequently examined under the microscope, also frequently exposed to sunlight, and at times to the powerful rays of the electric arc in the photomicrographic work, but were in nowise faded or injured after a period of two years from the time of preparation.

In the micrographic work the most painstaking care was employed in every detail of the work. Achromatic objectives of various power with compensating eye-pieces in different combinations were used so as to produce different magnifications up to 1,400 diameters. Oil-immersion lenses were not used, as the above combination secured the greatest possible depth under such high powers. The time of exposure varied from a few seconds in the low power photomicrographs to five minutes in some of those magnified 1,400 diameters.

*Material.* Some twenty brains were secured, and sections made from various parts of them all and studied in connection with this article. Three brains of cases electrocuted at Ossining, and in which the autopsy occurred immediately afterwards and the material placed in various fixative media, were secured. The photomicrographs representing the practically normal brain histologically in the first part of this article were taken from one of these brains, marked A (Plate I, Fig. 1), a man thirty-six years of age who had been confined in Sing-Sing State Prison for over two years and leading the regular routine prison life during that period. The autopsy was held immediately after death by electrocution. The body was well nourished, and no pathological condition of the central nervous system or other organs was found. Small blocks, .3 to .5 c. c. in thickness, from various convolutions of the brain were placed in the various hardening or fixative agents described above, and the brain itself immersed in 95 per cent alcohol. The blocks were subsequently imbedded in paraffin, sectioned serially, stained, differentiated, and mounted as already described.

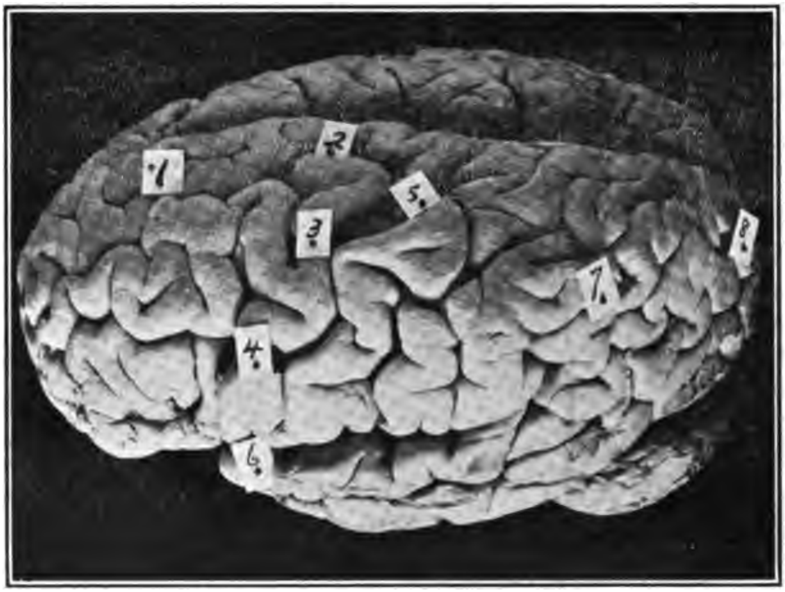


Plate I, Fig. 1.

Brains from three cases of dementia paralytica were also secured and studied. Sections from the one marked B (Plate X, Fig. 29) were used to illustrate this article in its pathological portion. The paralytic dement from which brain B was taken was a lawyer, had a collegiate education, was single, no history of syphilis, and family history negative. Was native of the United States. The disease began at about the age of twenty-eight years with the usual change of character, followed by grandiose ideas and maniacal excitement. Asylum treatment for over a year caused his symptoms to clear up sufficiently so that he was discharged. For nearly two years he was in a fairly quiet condition. He then broke out in a long period of excitement, lasting over a year, followed by terminal dementia lasting for one and a half years before death, which latter was uncomplicated. Duration of the disease was thus over five and a half years, during which period there was a remission lasting two years, in which he did not require asylum treatment. There was contracture of both arms and legs for three months preceding death. Mentally he was a little brighter during that time. At death the body was immediately placed upon ice, and the autopsy performed fifteen hours later. The body weighed 78 pounds at death and was flexed. Two small cavities, each the size of a pea, were found in the apex of the right lung. The lymphatics were somewhat enlarged. Heart was atrophied. There was meningeal thickening. The ependyma in the posterior part of the floor of the fourth ventricle was very slightly granular. The membranes were anemic, and there was no edema or fluid. The convolutions, especially in the region of the central convolutions, are widely separated from one another; but, as will be described later, this is largely due to mechanical causes in subsequent fixation. In addition to these brains over a dozen were secured from other sources, mostly from the New York City Morgue, of persons who had committed suicide or died suddenly from accident or homicide, with autopsy performed within a few hours after death.

*Normal Cortex.* The descriptions and plates herein contained, and illustrating the normal human cortex, are not intended to give the idea of being in any way a complete exposition of such a vast subject; but it has been the aim to give photomicrographs with accompanying descriptions of typical sections and cells from various regions of the cortex, as a basis of comparison with sections taken from corresponding regions of the cortex in a case of dementia paralytica. Neither is this contribution intended to be in the nature of an atlas, being much too limited in scope for such a work; but to illustrate as far as practicable the structures found in what can be considered a fairly normal brain, with the conditions found in a brain of one dying from dementia paralytica. Various regions of the normal cortex from brain A, one of the electrocuted cases (Plate I, Fig. 1), with its accompanying plates will be first described, and then will follow a like description of the corresponding region with its accompanying illustrations of the brain of the case of dementia paralytica, brain B (Plate X, Fig. 29). Plate I, Fig. 1, above referred to, shows the left hemisphere of Brain A, natural size; a normal brain, with well marked convolutions and sulci. The small pieces of cardboard with their contained figures show the particular point from which the subsequent blocks were taken, and also illustrate the method of exact localization of blocks described under the heading of technique.

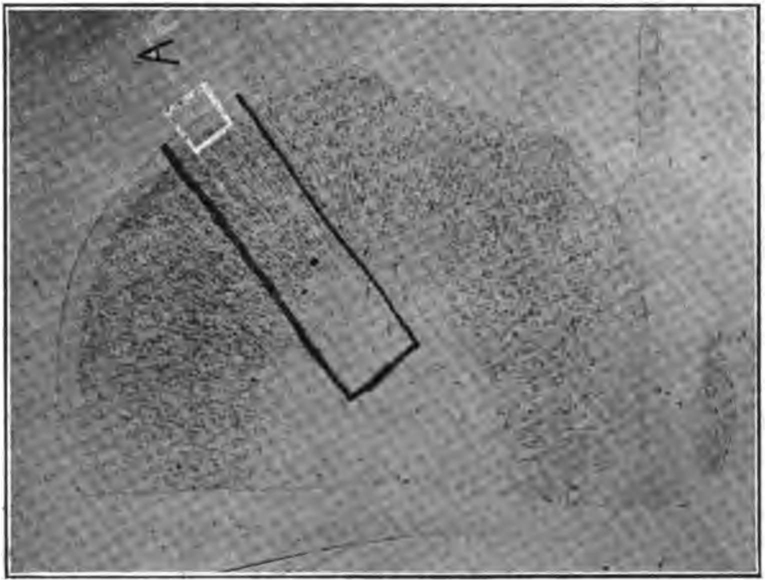
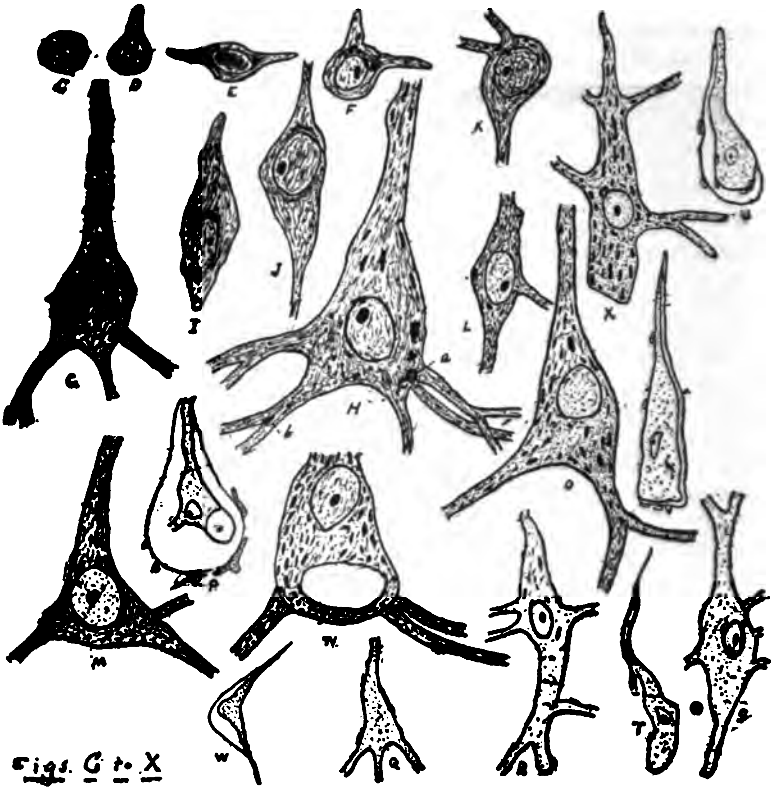


Plate I, Fig. 2.

Frontal Region.—Turning now to the several cortical regions, various portions of the first, second and third frontal convolutions were examined in different brains, and typical of this region is the section shown in Plate I, Fig. 2, which, with minor modifications, is similar to that of all portions of this region, and will now be described in detail. The block from which this section was taken comprised a part of the external surface of the first, or superior frontal convolution, including the entire width of the gyrus for a distance of .4 cm., and taken from the position shown by the figure 1, Plate I, Fig. 1. The gyrus at this point measures .6 cm. in width, so that the actual size of this section is .6 cm. in width by .4 cm. in depth, and it is 6 2-3 microns in thickness.

The block at autopsy immediately after death was placed in Van Gehuchten's fluid, where it remained for twelve hours, when it was placed in alcohol 95 per cent until ready for use. It was then imbedded in paraffin, sectioned serially with a Minot microtome, stained for two minutes in warm methylene blue, decolorized in alcohol, cleared in xylol, and mounted in xylol-damar. Plate I, Fig. 2, referred to above, shows the section under a magnification of 14 diameters. The section has been broken in manipulation, but shows the general striated arrangement of the cells radiating outward from the white medullary center to the surface; also the layering of the cortex. The strip of cor-



Figs. G to X

tex outlined in ink on the plate at *a* is that portion shown in Plate II, Fig. 3, under a magnification of 100 diameters. The outer pale layer, the thicker cellular layers, and the inner medullary white portion can easily be differentiated even with this magnification.

The outer layer is found to be fairly uniform in thickness in this section, but we will find later that there may be a considerable variation, not only in the thickness of this layer, but also of the other layers, and consequently of the entire cortex. The radial arrangement of the cells from the medullary white substance is especially well shown under this magnification, and a more general and comprehensive view of both the normal arrangements and of derangements and disturbances due to pathological changes can be made out under this magnification than by higher powers in which but minute areas are seen. These latter high magnifications are of course also necessary for the complete study of all the details of such variations or pathological changes. Plate II, Fig. 3, magnified 100 diameters, is that portion of the section outlined in ink in Plate I, Fig. 2 at *a*. Decolorization has been carried on here to such a degree as to make many of the cells appear somewhat pale and washed out, and to cause a complete decolorization of many of the neuroglia cells. This, however, admits of a better study of the arrangement of the internal structure of the cells, as a too deep colorization does not sufficiently differentiate the chromophilic granules and network. The outer surface of the cortex is here seen to be smooth and regular. Immediately beneath is the first or superficial layer, pale in color, .25 mm. in thickness, and characterized by a paucity of cells irregularly arranged and appearing upon a colorless background, which we know from sections treated by the Weigert method to be made up of large numbers of densely packed nerve fibers and processes. In the plate are seen for the most part neuroglia cells, but under a high power here and there nerve cells are found, rounded, spindle-shaped, or polygonal in form, with a nucleus filling up almost the entire cell-body, and with no visible dendritic processes, or at most but one or two extending vertically or at right angles to the surface of the cortex. The cell-body contains but a small mass of chromophilic substance, usually forming a narrow band about the nucleus with thickenings where the dendritic processes are given off. The cells with no visible processes of course may have such extending in a direction outside of the plane of the section. Fig. C shows one of these cells containing no visible processes, with a rounded cell-body, a large rounded nucleus almost filling up the same and surrounded by a uniformly thin band of chromatic substance. Fig. D shows another cell from this same region with a single proces vertical to the surface of the cortex. Here again the large rounded nucleus almost fills up the cell-body and is surrounded by a thin band of chromatic substance thickened at the point where the dendritic process is given off, and extending into the same for a short distance. Fig. E shows a similar cell, but with two processes extending in a nearly horizontal direction. The thin chromatic band is here thickened at both extremities. The greatest diameter of the cell in Fig. D is vertical, while in Fig. E it is horizontal, in Fig. C of course being practically uniform. Occasionally cells are seen with one process vertical and the other horizontal, as in Fig. F, this being polygonal in shape. The nuclei in all of these cells are well defined, large, and contain a small amount of chromatin in the form of a poorly differentiated network, and at some point containing a distinct nucleolus. According to Nissl's classification of nerve cells these would be known as karyochrome nerve cells. The neuroglia cells are smaller, rounded or oval in shape, contain a nucleus, and are much more numerous in this layer than the comparatively scarce nerve cells. The second or pyramidal cell layer, to simplify the layering of the cortex to the greatest extent, as previously described, is made to include both the large and small pyramidal cells. This measures in the plate (Plate II, Fig. 3) 1.40 mm. in depth. This layer thus includes the large and small pyramidal cell layers and the ganglionic cell layer of Hammarberg, and the large and small pyramidal cell layers of Golgi and of Cajal. (See Table I.) Hammarberg (1895) in his description of this region states that the layer of small pyramidal cells does not form a distinct layer, but gradually passes over into that of the third layer of large pyramidal cells, so that the two in his division make a layer .80 mm. in depth. He further states that the under border of the third layer is difficult to determine, so that there is no distinct fourth layer. This fourth layer is then described as containing small pyramidal cells less thickly distributed than in the layer above, intermingled with which are some smaller irregular cells, giving the appearance of a region poor in cells; and he notes further that in many places the difference is so slight that it does not make a separate layer. His fifth layer is described as a .70 mm. thick ganglion cell layer, made up of somewhat larger and more closely packed pyramidal cells. These layers,—second, third, fourth and fifth layers of Hammarberg, are all included in this second layer of the writer, and in this section measures about 1.40 mm. in thickness. It is made up of pyramidal cells varying in size in all parts of the layer, so that in any region, excepting a band below the middle portion to be described later, pyramidal cells of different size may be seen in close proximity.

As a rule, however, the larger pyramidal cells are found mostly in the deeper portion of the layer. But here as elsewhere are to be seen the small pyramidal cells interspersed. There are also to be seen some rounded and irregular cells throughout this layer. Just below the middle of this second layer is seen a belt some .30 mm. in width, in which small pyramidal cells decidedly predominate and containing but few larger and medium sized pyramidal cells. This region is comparable to the fourth layer of Hammarberg, but as it contains no elements differing from the region above and below, but simply varies in the relative number and size of the same, it does not seem to the writer worthy the designation of a separate layer with the resulting increase in the number of layers. Both above and below this belt large and small pyramidal cells are intermingled; the large pyramidal cells increasing in proportion from above downward. Fig G (G of Plate II, Fig. 3) shows the details of structure of one of these small pyramidal cells under a high magnification (x 1,300. No. 3 ocular, 1-12 in. oil immersion objective, Leitz). By focussing in different planes the large, broad, apical, dendritic process is seen extending vertically towards the surface of the cortex. It contains chromophilic granules arranged with the long axis parallel to the axis of the cell. The cell-body is in the form of a pyramid with rounded sides. The large nucleus is irregularly oval with the long axis parallel with the long axis of the cell. The nucleolus is centrally located and surrounded by the slightly stained nuclear contents. From the lower portion of the cell-body are given off three dendritic processes, one of which subsequently branches into two processes, at which point a mass of chromatin is seen, one of the so-called "wedges of division" (Versweigungs-Kegln) of Nissl. These dendritic processes contain no chromophilic granules, but at the point where they are given off from the cell-body are found aggregations of chromophilic granules which also surround the nucleus on either side, their long diameter extending in general parallel with the long axis of the cell. Plate II, Fig. 4, is a photomicrograph of this same cell as stated, indicated by the letter G in Plate II, Fig. 3, and shown in Fig. G of the text as already described. In order to secure a focus by which the cell processes could be seen even faintly the nucleus and nucleolus are almost entirely out of the plane of focus, the latter showing faintly however. Immediately about the nucleolus there is a slightly pale area, a portion of the nucleus. The contents of this cell-body itself present for the most part the appearance of a very dark, almost homogenous, intensely stained chromatic substance, and gives only a partial idea of the real structure and arrangement of the granules as seen in Fig. G. The contour of the nucleus cannot be distinctly made out, nor the relative position of the nucleolus in the same. The individual chromophilic granules cannot be distinguished excepting at the base of the right lateral dendritic process. The apical dendritic process appears as a mere shadow, owing to its being out of the plane of focus. The basal dendritic process show almost as well as in Fig. G, excepting the branching of the one at the left which is but faintly indicated. The small mass of chromatin at this point—the so-called "wedge of division," of Nissl—is also but faintly indicated. The general contour of the cell, points from which the dendritic processes are given off, and position of the nucleolus are accurately represented. Had a plane been selected showing the nucleus accurately some of the processes would have been entirely out of focus. Other cells adjacent to the one just described are seen to be less deeply stained and the chromatin is seen in the form of larger and smaller indistinctly rounded or elongated granules. It will thus be seen that there are limitations, and serious limitations too, in the use of photomicrographs, especially under high powers, in such a study as this. It would require several photomicrographs at various planes to bring out

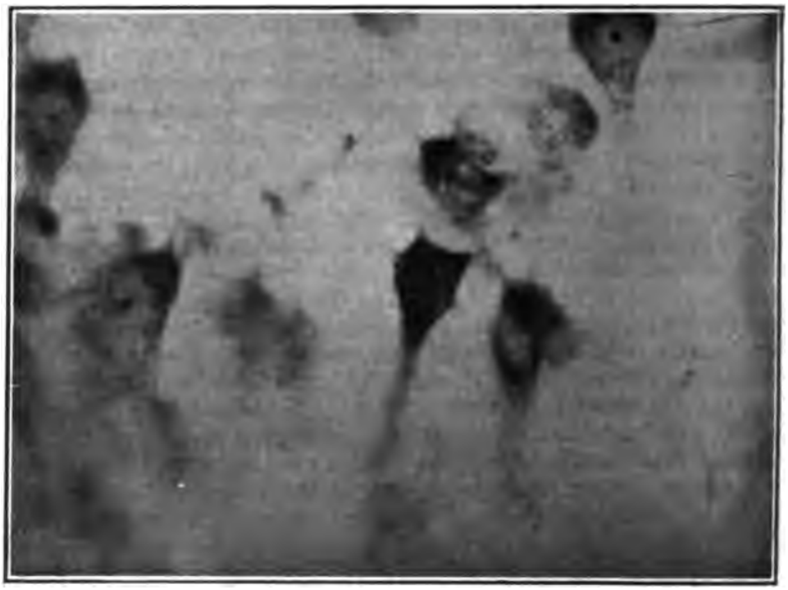


Plate II, Fig. 4.

all the details shown in the single Fig. G, which of course is a composite taken by focussing in all planes under a magnification of 1,300 diameters, and showing certain, but not all, details of the different planes. Of course outlines are not so distinct in the real cell as must necessarily be made to appear in a drawing, and this latter might be compared to a dissection in which certain parts are brought out prominently at the expense of the normal appearance of the whole in life. No one would hesitate to say, however, that Fig. G gave a much better idea though not an exact representation of the cell, than Plate II, Fig. 4, and that drawings are essential accompaniments of such plates in the full elucidation of a subject of this kind. These cells are known as stichocrome nerve cells of the somatochrome class. Fig. H shows one of the large pyramidal cells (the largest to be seen in the plate) under the same magnification as in Fig. G (x 1,300. No. 3 ocular, 1-12 in. oil immersion objective, Leitz). The internal structure of the large pyramidal cells is found to be made up of distinctly stained chromophilic substance in larger and smaller irregular granules, some so fine and closely arranged as to completely fill up the cell-body, thus presenting a uniform field of closely packed minute granules without any special relation to one another. In other cells the granules are coarser and present a somewhat parallel arrangement about the nulceus and extending up into the main apical process for some distance and also into the basal lateral processes in the same general parallel manner. In other pyramidal cells there is a fine chromophilic network with larger and smaller aggregations of chromatin as nodes or enlargements of the network. In this figure the cell-body is pyramidal in shape with rounded contour merging above into the large apical dendritic process, which is finally lost to view as it enters another plane. Below at the base, both to the right and left, are given off several dendritic processes, some of which are seen to branch a short distance from the cell-body. The large, slightly oval nucleus is centrally situated somewhat nearer the base of the cell than the apex, and contains a large, eccentrically placed nucleolus, surrounded by the pale, slightly strained, for the most part homogenous, nuclear protoplasm. Within the cell-body about the nucleus and extending into the apical process, and to a less extent into the basal dendritic processes, are chromophilic granules of rounded or elongated shape, and having a tendency to be arranged in groups in places. Especially at the point where a dendritic process is given off is frequently to be found a mass of these granules. Often a wedge or cap of chromophilic substance is seen at the point where a dendritic process is to be given off, as at *a*, or where a process divides into two branches, as at *b*, the latter being one of the so-called "wedges of division" of Nissl. The granules vary in size and shape and are arranged in general in a direction parallel to the long axis of the cell-body and of the long axis of the dendritic process when extending into the same. About the nucleus they are often arranged parallel to its wall. Five dendritic processes are given off from the base of this cell, three of which are seen to divide into two branches each; at *b*, showing one of the so-called "wedges of division" of Nissl, referred to above. Plate II, Fig. 5 is a photomicrograph of this same cell indicated by the figure H in Plate II, Fig. 3, and already described and shown in Fig. H; here under a magnification of 1,400 diameters. The body of another somewhat smaller pyramidal cell is seen just behind the apical dendritic process of this cell and part of it almost in

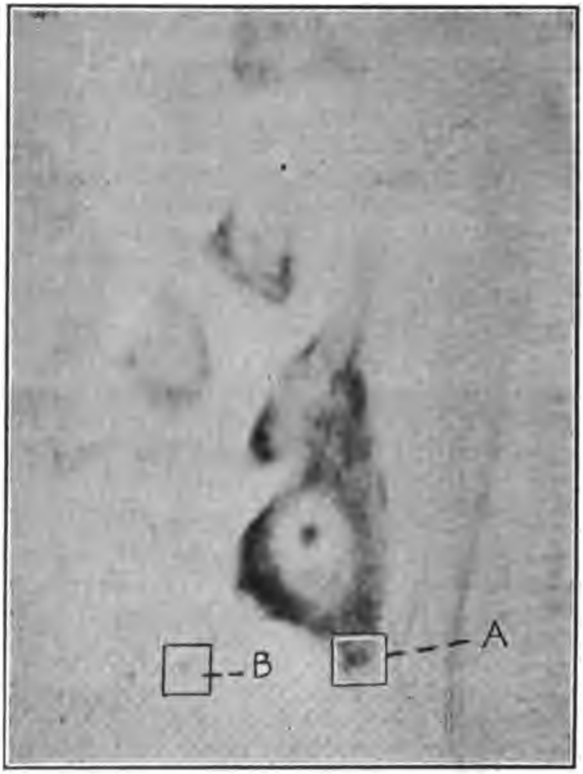


Plate II, Fig. 5.

the same place, so that it is difficult to differentiate the two. It will be seen that the nucleus and nucleolus are in good focus, but that the basal dendritic processes, excepting at their origin from the cell-body, can scarcely be distinguished. The shape of the nucleus, size and position of the nucleolus, and arrangement of the chromatin within the same is as well shown as in the figure. Also the shape and contour of the cell-body. The dendritic processes, however, as mentioned above, are out of the plane of focus, so that only a very hazy, indefinite indication of the beginning of three of them can be made out. The large one at the left can scarcely be determined at all. The more central one at the left is faintly outlined, and at the point of bifurcation the so-called "wedge of division" of Nissl (B) is seen, but the division itself cannot be determined. Of the three processes given off from the base at the right the beginning of the one most centrally located can be very faintly seen and a faint outline of the other two, appearing as one in the photomicrograph is seen. The apical dendritic process is so merged with the body of the cell just below it that it is difficult to determine its outline. The cell protoplasm is more differentiated however than in the preceding plate (Plate II, Fig. 4). Here chromophilic bodies are seen at the base on the right where the dendritic processes are given off, indicated by A both here and in Fig. H. Chromophilic granules are also seen fairly well indicated to the right and above the nucleus, in larger and smaller bodies without sharp outlines, which is the condition actually found in the cell. They are not sharply and definitely outlined as shown in Fig. H, which in that respect is not accurate, but merely diagrammatic. At the left and below the nucleus in the basal portion of the cell-body these chromophilic bodies are so closely aggregated as to give almost a homogenous dark appearance to that portion of the cell, and it is only by carefully focussing in various planes that the chromophilic bodies are made out. Here again the photomicrograph without the accompanying drawing would give one only a partial knowledge of the structure of this cell. Cells of this type are to be classified as large stichochrome nerve cells of the somatochrome class. Interspersed among these larger and smaller pyramidal cells in this layer are some small irregular and rounded cells consisting of a large nucleus containing a nucleolus, and surrounded by a cell-body containing in the smaller cells a very narrow rim of finely granular chromophilic substance, in some places so narrow as to be scarcely discernible. In other cells the cell-body is much more developed surrounding the nucleus and giving off several protoplasmic processes which go out in various directions. Neuroglia cells similar to those of the first layer are found interspersed among the nerve cells in all parts of the section. Small portions of capillary blood vessels are shown as at I in the plate (Plate II, Fig. 3, I) where the vessel is seen to divide into two branches below, and also at 2 (Plate II, Fig. 3, 2), showing a single small capillary extending only for a short distance in this plane. The walls are seen to be exceedingly thin, made up of a single layer of nucleated cells, and containing red blood corpuscles. The walls of these blood vessels stand in marked contrast to the thickened and tortuous walled vessels found in the cases of dementia paralytica later to be described. Below this is the third, or spindle cell layer about 1.20 mm. in thickness in this section. The spindle cells in this layer contain a large nucleus, with a distinct nucleolus, but no nuclear net work could be made out. The nuclei in many of them present the appearance of being too large for the cell-body, so that often, as in Fig. I, the cell-body seems to be bulged out on one side to accommodate the large nucleus, giving an eccentric form, and resulting in a spindle with one side very prominent and the opposite side quite flattened. The chromatic substance is arranged in no definite network and contains no distinct chromophilic bodies, but consists of minute particles closely aggregated about the nucleus, especially at each pole and found in a lesser amount and in variable quantities in the remainder of the cell-body. In Fig. J the nucleus is situated eccentrically at one end of the cell-body, so that most of the other cell contents are at the opposite end with a narrow band of closely aggregated chromophilic substance surrounding the nucleus at both ends. The nucleus in this cell is more elongated and oval in shape, with distinct nucleolus and otherwise pale, slightly stained contents. Other cells as Fig. K are more or less irregular in shape, in this case having the form of an irregular inverted pyramid containing a large rounded nucleus near the base which is surrounded by a narrow band of dense chromatic substance. About this is an irregular chromatic network containing nodal thickenings at some points. The nucleus contains a distinct nucleolus, but otherwise pale and but slightly stained contents. A large dendritic process is seen given off below and two dendritic process go off from the base and to one side. Fig. L shows another irregular shaped cell with a dendritic process given off from one side in addition to those from each end. The large oval nucleus with distinct nucleolus and otherwise pale contents is centrally placed. Considerable chromatic substance is seen in the larger cell-body with distinct granules, forming an indistinct network at the point where the lateral dendritic process is given off, on the opposite side and also in the cell-body and at the base of each of the vertical processes. Plate III, Fig. 6, is a photomicrograph, at a magnification of 1,400 diameters, of a group of the spindle and irregular cells indicated by the letter I in Plate II, Fig. 3.

As will be noted, almost all the forms shown in the above described figures are to be seen here. The cell lettered I, is similar in shape and structure to that of Fig. I, including the bulging out of the nucleus to one side, and showing an aggregation of chromatic substance in the form of a nuclear cap at both poles of the nucleus. An irregular network of chromatin is seen within the nucleus surrounding the nucleolus. But little chromatic substance, in addition to the two nuclear caps above described, is to be seen in the small cell-body. Again the cell indicated by the letter J is very similar to that seen in Fig. J, the nucleus being at one end of the cell-body near the base of that polar dendrite. The nucleolus is situated at the lower portion and to the left in the nucleus and with but little faintly stained chromatic substance surrounding it. About the nucleus in the cell-body and extending into both dendritic processes is to be seen considerable chromatic substance arranged in an indefinite network. The cell lettered K is irregularly pyramidal

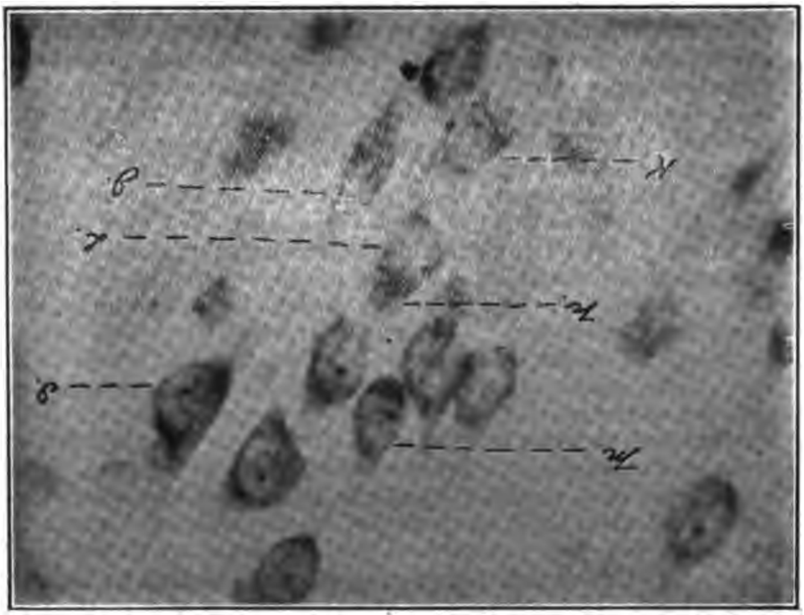


Plate III, Fig. 6.

in shape, approximating to that seen in Fig. K. The nucleus is very large and almost entirely fills up the cell-body. The nucleolus has the appearance of being without the nucleus, but is within the same adjacent to the wall at this point. A small amount of chromatin is seen in the lower part of the nucleus, otherwise it is for the most part pale. A process is seen given off above as the apical dendrite at the base of which are some few chromophilic granules.

Two basal dendritic processes are given off, one to the right and projecting almost vertically downward, and the other to the left and almost horizontal in direction. At the base of these processes, especially the one to the left, are seen well marked chromophilic granules. Otherwise the cell-body is practically entirely taken up by the nucleus. The cell indicated by the letter L is somewhat similar to that of Fig. L in regard to having two polar dendritic processes, one of which coming off laterally here on the left side and only faintly indicated, being in a lower plane. The nucleus here also is seen to occupy the greater portion of the cell-body, the nucleolus being crowded over almost to the edge of the same. The nucleus contains only a slight amount of chromatic substance in its upper portion. Nuclear caps are seen above and below the nucleus in the cell-body, the one above being the larger, and surrounded by some small chromophilic bodies extending up into the dendritic process. A very slight amount of very palely colored chromatic substance is seen below the nuclear cap in the lower dendritic process. The lateral dendrite when seen in focus is found to contain a slight amount of chromatic substance more abundant at the base. The cell lettered M is regularly spindle in shape with two polar dendritic processes, the lower one not in the plane of focus, and the upper one only partially so, and there are no lateral processes. The nucleus is not in distinct focus, so that its outline is indistinct. The nucleolus is seen at the lower part of the nucleus, and to the left side surrounded by a rather pale indefinite chromatic network. Below this is seen some dense chromatic substance in the form of a nuclear cap. The nucleus really occupies almost the entire body of this cell extending from the dark mass below which has the form of an irregular nuclear cap, to the base of the upper dendritic process where there is a smaller nuclear cap of chromatic substance. A small amount of protoplasm above and below the nucleus in the cell-body extends for a short distance into the dendritic process from these nuclear caps. The other cells seen in this plate are similar in appearance and structure to those already described.

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(Plate illustrations referred to and not found will appear in the November issue.)

(*To be continued.*)

# Periscope.

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### NOUVELLE ICONOGRAPHIE DE LA SALPETRIERE

(Vol. 16, 1903, No. 1, January-February.)

1. Chronic Hypertrophic Acrocyanosis. M. Péhu.
2. A Case of Progressive Facial Hemiatrophy. Calmette and Pagès.
3. Acquired and Progressive Chronic Trophoedema. J. Sicard and Laignel-Lavastine.
4. A Case of Macrodactylia. F. Lejras.
5. Macrodactylia. Cayla.
6. In Regard to the So-Called Sense of Position. Claparede.
7. Les Urologues. Henry Meige.
8. A Note Upon Three Designs of Jordæns. Jean Heitz.

1. *Chronic Hypertrophic Acrocyanosis.*—The pathology of the extremities, acropathology, is yet a limited field in medicine. The types at present known are Raynaud's Disease, L'Osteoarthropathie hypertrophiant pneumonique of P. Marie, and Acroparesthesia of Schultz. The characteristics of these processes are the localization and the sensory vasomotor or trophic type of the symptoms. It is not always possible to differentiate sharply each variety from the others on account of their complexity. In addition to Raynaud's disease, there are three main types in which asphyxia is constant and chronic; they are acrocyanosis with sensory symptoms, with atrophy, and with increase in the volume of the soft parts. The author of this paper, after giving a literature resumé of cases previously described, gives one of his own which evidently belongs to the third class. Case.—Young man, twenty-five years of age, neuropathic history, alcoholic, tuberculous, presented during a period of ten years' intermittent attacks of cyanosis of hands and feet. The asphyxia became in time permanent, and was accompanied by pain so severe that he had to give up his daily work. In addition, the hands increased in volume without any corresponding lesion of the bone (radiograph). There were never any sensory disturbances and no attacks of local syncope. The following were considered in the differential diagnosis: L'osteoarthrop hypertrophiant pneumonique of P. Marie, acromegaly, acroparesthesia, erythromalalgia, and Raynaud's disease.

2. *Facial Hemiatrophy.*—A case of progressive facial hemiatrophy in a young girl, fifteen years old. There are ewo theories to account for this disease: First, primary atrophy of the subcutaneous tissue; second, a lesion in the facial nerve, sympathetic, or the trigeminal, the so-called nervous theory. Brissaud's theory seems more reasonable. "In the presence of a hemilateral affection, involving the seventh, fifth and twelfth nerves, it is natural to think of a lesion in the cerebrospinal axis. "This paper is illustrated by some remarkable photographs.

3. *Chronic Trophedema.*—A classification of edemata based upon the evolution of the symptoms, and not upon the etiology or pathogenesis, seems with our present knowledge the most simple and effective. Henry Meige, in a series of studies, has described a type to which he has given the name of chronic trophoedema. The following divisions and subdivisions are at present recognized: 1. Acute Edemata: (a) infectious, (b) neuropathic; 2. Hysterical Edemata; 3. Chronic Edemata: (a) depending upon various chronic diseases of the heart, lungs, nervous system, etc.; (b) chronic edema of elephantiasis; (c) tropho-edema (Meige). This paper is based upon a case of chronic acquired tropho-edema. Girl,

twenty-seven years of age, neuropathic heredity; as a result of a trauma of the left foot a painful edema appeared, localized at the site and a little above the contused region. This edema was at first transient, but gradually became permanent, and progressively involved the leg, thigh, buttocks, of the same side. Process was a slow one, taking in all two years.

4. *Macrodactyly.*—A case of local gigantism in a child thirteen years old. The median finger of the left hand was enormously enlarged. There was no hereditary history. This finger measured 15 cc. in length, 12 cc. in circumference in the middle of the first phalanx; finger was amputated. The article is illustrated by radiographs and photographs.

5. *Macrodactyly.*—A further case of macrodactylia in a boy seventeen years old.

6. *Sense of Position.*—A discussion of Bonnier's paper on the sense of position, which appeared in No. 2, 1902, of this Journal. (Not adapted to abstract.)

7, 8. Historical and literary notes on medicine in art.

SCHWAB (St. Louis).

(Vol. 16, 1903, No. 2, March-April.)

1. Syringomyelia; Arthopathy of the Shoulder (Muscular Atrophy and Thermoanalgesia of a Transverse Type). E. BRISSAUD and BRUANDET.
2. An Old Lesion of the Red Nucleus. Secondary Degeneration. PIERRE MARIE and GEORGES GUILLAIN.
3. A Case of Late Hereditary Syphilis of a Cerebrospinal Form. RICHON.
4. A Case of Symmetrical Sclerosis of the Occipital Lobe. L. MARCHAND.
5. A Case of Progressive Myopathic Atrophy, with Sensory Symptoms. M. LANNOIS and A. PAROT.
6. Enormous Deformity of the Right Tibia (Forreau de Sabre) in a Tabetic. J. SABRAZES.
7. A Case of Phocomelia and Hemimelia. P. HALBRON.
8. Some Examples of Edemata in Art. HENRI MEIGE.

1. *Syringomyelia.*—Man, forty-nine years, with tuberculosis of the lungs and a scapulo-humeral articulation very much deformed and augmented in volume. The diagnosis of syringomyelia was based upon the arthropathy, muscular progressive atrophy of the left upper extremity, and a dissociation of sensibility localized in both arms. The man died of tuberculosis. Microscopical examination of the cord showed typical appearances of syringomyelia. The shoulder joint showed trophic changes of a very interesting nature. The articular capsule had disappeared together with the ligaments. The long head of the biceps was absorbed; its insertion in the glenoid cavity could not be recognized. Villous vegetations filled the joint. In the muscles of the subscapular, supra, and infraspinous regions ossiform nodules could be demonstrated.

2. *Lesion of Red Nucleus.*—An old lesion found in the red nucleus and causing secondary degeneration of various nerve tracts gave the subject matter of this paper. On account of the rarity and limitation of the lesion, a study of sections above and below it seemed to promise interesting results. The subject of this study had infantile cerebral palsy, from which a left hemiplegia remained. At the age of twenty-seven years he became infected with syphilis, death occurred at forty-nine, with progressive dementia. No cortical or meningeal lesions could be demonstrated. On the left side, exactly in the red nucleus, a lesion was found. It was either an old tubercle, which had passed into the sclerotic state, or an old limited focus of softening. Posteriorly and anteriorly to the red nucleus the structures are normal, showing the sharp limitation of the lesion. It could be compared in fact to a veritable experimental destruction of the red nucleus.

Cross sections above and below the lesion showed very interesting fiber degenerations, the most important of which are degeneration of the cerebellar peduncle, the central tract of the calotte, and the posterior longitudinal bundle. Very excellent plates illustrate the article.

3. *Tardy Syphilis.*—This is a very careful histological study of a case of tardy congenital syphilis in which the lesions were localized chiefly in the cerebrospinal system. The etiological diagnosis could only be made from a study of the lesions. Boy, age eleven years, without visible lesions or stigmata of inherited syphilis. Fistulous lesion of the parietal bone accompanied by Jacksonian epilepsy. Unsuccessful surgical intervention. Persistence of the epileptiform convulsions, accompanied by hemiplegia, with contracture. Progressive decline in intellect, deafness, blindness. Cachexia and terminal tuberculous infection. Autopsy: Fibrous meningo-encephalitis, basal gummatous meningitis, cavity formation in the cerebellum, infiltration of spinal meninges. Examination of the skull showed evidences of an extension of the fibrous encephalitis into the bony substance. The evolution of this condition is thus summarized: The brain already structurally deformed, with probably a certain degree of hydrocephalus, a condition not rare in congenital syphilis, became the seat of a gummatous neoplasm. A gumma arising from the periosteum, or osseous tissue, perforated the inner table and attacked subjacent tissue, producing adhesions. The meningeal encephalitis developed slowly at first and led to a sclerosis of the hemispheres, causing the symptoms of cortical irritaiton, that is the epilepsy, and later the hemiplegia. The lesions then became more diffuse, extended to the cord, the base of the brain and to the white matter of the cerebellum. The specific nature of these lesions is very evident on account of the evolution of the process and their anatomical character, as well as by the existence of a veritable syphilitic stigma in the cerebral malformation.

4. *Sclerosis of Occipital Lobe.*—This case is interesting not only because it confirms the previous work on visual localization, but because it determines the group of symptoms which follow a symmetrical sclerosis of the occipital lobe. The slow course of the disease, and the definite lesion leading to a complete destruction of the occipital lobe add particularly to the interest of the case. Girl, nineteen years old; epilepsy; almost complete blindness; central vision for light alone remains, but the patient cannot recognize objects. The eyes are normal. Patient died in status epilepticus. Autopsy: The external and internal surfaces of both occipital lobes are covered by a membrane penetrating into the cerebral substance. The pia mater is adherent. The convolutions are much atrophied. The internal surface presents the same appearance. Microscopical examination shows sclerosis with numerous foci of softening and a great increase in neuroglia. The preservation of a visual field to light around the point of fixation in this case is of great interest, and seems to uphold the theory of V. Monakow, to the effect that the fibers corresponding to the macula are distributed in the whole extent of the cortical field of vision.

5. *Myopathic Atrophy.*—The distinction between muscular atrophy of myopathic and nervous origin is gradually being given up, and an intermediate variety, which includes many irregular forms, is being substituted. Various transitional types belong here. Such a one forms the subject of this paper. Clinical résumé: Man, thirty-eight years old; no family disease; neuropathic heredity; traumatism at seven years; first appearance of symptoms at ten years by lancinating pain and paresis of the muscles. Slow progressive development with a persistence of the pain and pseudo-hypertrophy of the lower extremities; muscular atrophy in all four extremities, symmetrical in respect to nerve root distribution and almost total in respect to segmental distribution; face not involved. Pseudo-hyper-

trophy of lower extremities, tendon contractures, abolition of tendon reflexes; almost complete disappearance of electric excitability, subjective and objective sensory disturbances.

6. *Tabetic Arthropathy.*—Cases of tabes with evidence of syphilis other than the metasyphilitic nervous lesions, are always of great interest. A number of cases belonging to this variety are increasing rapidly. The case here described is that of a man, forty years old, symptoms of tabes appearing at thirty-eight, as follows: Gastric crises, ocular symptoms, diplopia, Argyl-Robertson pupil, abolition of knee reflex. No history of acquired syphilis. Evidence of hereditary syphilis is present in the characteristic dental malformation and in a sword-like deformity of the right tibia (En Fourreau de Sabre). The character of the tabes in this case is very similar to that which is found in infantile tabes depending upon hereditary syphilis.

7. *Phocomelus.*—Description and photograph of a case of Phocomelia and Hemimelia.

8. An Art Study. SCHWAB.

### JOURNAL DE NEUROLOGIE

(1903, VIII, No. 5.)

1. The Differential Diagnosis of Epilepsy. KOWALEVSKY.
2. Incipient Tabes. DE BUCK.

1. *Epilepsy.*—In a very complete review, the author compares the symptoms of epilepsy with those of apoplexy, hysteria, eclampsia and convulsions from other sources, the paretic seizure and vertigoes. Sketching the characteristics of epileptic psychical disturbances, he gives the chief points distinguishing them from acute mental symptoms due to other causes. He then considers the characteristics from a diagnostic and prognostic point of view, of epilepsies of different kinds with the special indications for their treatment. Of particular interest are his remarks on epilepsy of syphilitic origin.

2. *Incipient Tabes.*—The author exhibited at a meeting of the Belgian Neurological Society, the case of a man of thirty-one years, without alcoholic or syphilitic history, but acknowledging abusus Veneri, who presented the following symptoms: Fatigue in walking, slight dragging of the feet, with dysesthetic sensations in the lower limbs, slight diminution of the pain sense in the area of distribution of the second sacral segment, slight ataxia and Romberg, exaggeration of the patellar and arm reflexes, loss of the plantar, Achilles tendon, cremasteric and pharyngeal reflexes. His pupils reacted to light and accommodation. No change in the electric reactions, sphincters normal, constant genital irritation with tendency to priapism. The diagnosis of incipient tabes he bases upon the sensory troubles, and especially upon the loss of the Achilles tendon reflex, and quotes a number of authorities in support of this view of the case.

ALLEN (Trenton).

(Vol. 8, 1903, No. 6, March 20.)

1. Tuberculous Polyneuritis with Exaggeration of the Tendon Reflex in the Rabbit. D. DE BUCK.
2. Infantile Cerebral Palsy. BASTIN.
3. Tic. DE BUCK.

1. *Tuberculous Polyneuritis.* It is well known that in multiple neuritis, the tendon reflexes are occasionally found to be exaggerated. This has been accounted for in three ways: (1) As due to increased irritability of the centripetal fibers (the nerve not being totally degenerated); (2) as due to increased irritability of the muscular fibers, and (3) as due to the

action of the poison of the disease upon the nerve cells in the cord, increasing their irritability. The author, after reviewing briefly these theories, as put forward by different writers, describes his findings in the case of a rabbit affected with neuritis in the posterior extremities, in the course of a general tuberculosis, in which the tendon reflexes were exaggerated. The muscles of the affected limbs were practically normal, the nerves showed marked parenchymatous neuritis, and there was diffuse degeneration of the columns of the cord. The cells of the anterior horns, and of the spinal ganglia, on the contrary, were normal. These findings seem to dispose of the muscular theory, and to give little support to that of cell irritation, but point rather to increased irritability of the nerve fibers as a cause of the symptom. However, the author thinks that the question as to the cutting off of inhibition by the severing of the paths from the superior to the inferior nerve centers, is to be considered, on account of the degeneration found in the pyramidal tracts.

2. *Infantile Cerebral Palsy.*—A case, history and demonstration, bringing out nothing specially new.

3. *Note on Case of Tic.*—The demonstration of a case of involuntary movements of the facial neck and shoulder muscles. The author diagnoses the case as one of tic, and in connection with it discusses the pathogeny of this disease and of the allied conditions of myoclonus, chorea and athetosis. He seeks to explain the difference between true tic and the other conditions in question in the existence of two sets of cortico-spinal fibers, the pyramidal, or direct, and the extrapyramidal, or cortico-mesencephale-spinal. In true tic (or mental tic, as he calls it) the impulse passes from the psychomotor region of the cortex by the direct way. In other involuntary movements, on the contrary, it passes down by the extra-pyramidal tracts, or depends upon a reflex irritation radiating from the spinal cord. The author some years ago formulated the theory that the localization of the irritation giving rise to "clonic involuntary hyperkinesias" should be found in the basal ganglia or in the extrapyramidal motor fibers descending from them. Proof of the correctness of this has, in the meantime, been accumulating, he thinks, and he refers in this connection to the papers of Redlich, Probst, Rothman, Sorgo, and others, who have studied the results of lesions in this region.

(Vol. 8, 1903, No. 7, April 5.)

1. Circular Insanity and the Circular Forms of Psychoses. S. Soukhanoff and P. Ganouchkine (also see *Arch. de Neurologie,* May, 1903).
2. Medical Graphology. Mlle. D. Peppee.

1. *Circular Insanity.*—The author gives the results of a study of 86 cases of circular insanity from the Moscow Psychiatric Clinic, and draw the following conclusions. This form of insanity is nearly three times more frequent in women than in men; it constitutes 1.94 per cent of all the cases (4,434) received. It is twice as frequent as mania, and only one-third as common as melancholia. In its etiology, heredity is quite as important as in mania, in which it was found to play a rôle in 84 per cent of all cases. Two-thirds of all cases of circular insanity begin between the ages of sixteen and twenty-five years. In the majority of cases the disease is ushered in by depression. They then take up the question of the occurrence of psychoses in circular forms, and narrate some interesting cases, in some of which the disease, though showing alternating phases, was evidently due to organic changes in the brain.

2. *Medical Graphology.*—A discussion, from a diagnostic point of view, of the characteristics of handwriting. The author, a handwriting expert, makes certain suggestions as to conclusions as to character and state of

mind of an individual which may be drawn from a study of his or her handwriting.

(Vol. 8, 1903, No. 8, April 20.)

1. The Patellar Reflex in Neuroses. L. SCHNYDER.
2. Dolorific Assymetry. J. IOTEYKO and M. STEPHANOWSKA.

1. *Patellar Reflex.*—The author calls attention to the extreme difficulty in securing relaxation of the limb when examining the condition of the patellar reflex in nervous subjects, the patients usually seeming unable to keep from contracting either the extensor or the flexor muscle of the leg. He regards this as constituting a symptom of some value as indicating nervous irritability, and compares its method of production with that of the muscular rigidity found in myotonia congenita, in catalepsy, and the negativism of certain mental diseases, and in hysterical contraction. To illustrate his views he gives short clinical histories of ten patients suffering from neurasthenic, hysterical and mild mental symptoms, in whom this phenomenon was present.

2. *Dolorific Assymetry.*—It has long been known that there is a difference in general sensibility upon the two sides of the body, greater upon the right in right-handed persons, and upon the left in left-handed ones. Van Biervliet has shown that the proportion is usually as 10 to 9. Starting out from the above facts, and from the researches of Goldscheider and Von Frey, which seem to show proof that there are special nerve fibers, respectively for pressure, for pain, and for temperature sense, the authors carried on a series of experiments with a view of ascertaining if there is any difference in the acuteness of the pain sense upon the two sides. In their measurements they made use of the algesimeter of Chéron. Their experiments were performed upon 52 subjects, mainly university students, fourteen of this number being left-handed. The points chosen for pricking by the instrument were exactly symmetrical areas on the anterior surfaces of each forearm, just above the wrist. The following are the results obtained: Two of the subjects were practically insensible to pain. Of the remaining fifty, three persons showed greater sensitiveness upon the right side, but in two of these even, the result was doubtful. All of the remaining forty-seven showed greater sensibility to pain upon the left side, both right-handed and left-handed persons being included in the number. Taking the average of the elgesimeter readings for all of these, the sensibility of the two sides was found to be as 10 to 9 in favor of the left, conforming to the figures of Van Biervliet. Experimenting upon other regions (the finger tips, back of the hand, temples), the authors still found sharper pain sense in these cases always on the left. They make no attempt to explain the cause of the difference. C. L. ALLEN, Trenton.

### PSYCHIATRISCHE, NEUROLOGISCHE, WOCHENSCHRIFT

(Vol. 4, 1903, No. 48.)

*The Treatment of Epilepsy According to the Toulouse-Richet Method.*—Halmi and Bagarus. Fifteen cases of epilepsy which had been under observation in the hospital for some time were selected for the investigation. For ten months these patients were treated with 3-5 gms. of bromide daily, then followed two months without any medicinal treatment, at the end of which time the Toulouse-Richet method of treatment was applied for one month. During the first half of this period 3 gms. of bromide were given, when the dosage was reduced to 1.5 gms.; diet consisting of 2 litres of milk, 2 eggs, and for the men 750 gms., for the women 500 gms. of salted bread. Following this period of treatment they returned to the simple bromide treatment. In view of the great variation in the number of attacks monthly during treatment with simple bromides—in one case

19-31, in another 3-104 attacks, it would seem manifestly unfair to accredit a cure unless there was improvement in a large majority of the cases. Furthermore, in the nine months without change of treatment, nine cases showed spontaneous improvement. During the two months when the bromides were withdrawn the attacks increased in seven and diminished in three cases, and two patients died, but the total number of attacks fell to 273, or nearly one half of the total number in the two months when the bromides were withdrawn. Still, during six of the ten months in which the simple bromides were given, the total number of attacks was less than during the test month. Furthermore, a close study of the individual cases which seemed most improved reveals the fact that just as great improvement had previously occurred during the treatment with the simple bromides. In the final period of seven months the total number of attacks in the thirteen remaining cases rose to 324, but later fell to 219, in spite of the fact that the bromides were entirely withdrawn in five cases. Besides this doubtful effect in the number of the convulsions, this method of treatment had an appreciable sedative influence upon all psychical processes. The patients were less restless and exhibited a retardation which, in some cases, almost reached genuine stupor. In two cases it produced a fatal intoxication. In conclusion, the Toulouse-Richet method neither cures nor benefits, and greatly enhances the danger of bromism.

(Vol. 5, 1903, No. 1.)

The author (Waltenberg), after citing the theoretical and practical reasons for the abolishment of the "isolation cell" in the treatment of maniacal patients, presents the results accomplished during the past six years (1896-1902) at Lübeck, by the substitution of the constant watch, and the prolonged bath. He observes that during this period a greater degree of quiet prevailed, and there was a proportionate less demand for narcotics. The plotted curves accompanying his paper show the gradual fall of the percentage of women reported restless at night (11.3 per cent to 3.2 per cent), and a similar fall in the use of narcotics (3.6 per cent to 1.2 per cent).

(Vol. 5, 1902, No. 2.)

The Prognosis and Therapy of Intemperance. SAUERMANN-BONN.

The author calls attention to the increasing prominence of institutions for the treatment of inebriates, the reports of which record from 15-80 of recoveries, to the prominent rôle played by the "after treatment" in the third conference of the German inebriate institutions, and cites the method instituted at the Swiss Institution Ellikon by the establishment of a society of discharged patients for the purpose of mutual aid in maintaining abstinence. Their results are 70 per cent of recoveries from 1896-1900. His statistics relative to the prognosis are based upon 246 replies from members of the Red Cross Abstinence Society. He concludes (1) that recovery from alcoholic intemperance is synonymous with prolonged abstinence; (2) the frequency of defective heredity and a psychopathic constitution has led to the belief that a large number of alcoholics are incurable. The real basis for this belief lies in a lessened resistance to the action of alcohol; the drink custom and other accompanying influences only give opportunity. The patient cannot be cured of hereditary intolerance, he can only be freed from alcohol. Recovery from acquired intolerance is possible. (3) The necessity of prolonged abstinence and the great tendency to relapse demands prolonged attention after dismissed from treatment. (4) Secret abstinence societies are well adapted to exercise this extended cure. They not only prevent but lessen the danger of relapse. (5) Temperance abstinence societies should affiliate with public institutions for the treatment of alcoholics. (6) The fact that abstinence can be secured without institutional treatment will make it possible in many cases to shorten the time of treatment now prescribed (from six to twelve months). In spite of these results he believes that in the majority of cases institutional treatment is necessary. In this treatment, employment of the patients is essential for their betterment, and conduces to economy in their support at the institution. **Defendorf** (Middleton, Conn.).

### ARCHIVES DE NÉUROLOGIE

• (Vol .XV, 1903, No. 88, April.)

1. Historical and Critical. Physio-psychology of Women in Religious Orders; the Nuns of Port Royal (Fifth Series of Five Observations). Charles Binet-Sangle.
2. Upon Some Clinical Particularities of Facial Neuralgia and Its Treatment by Electricity. A. Zimmern.
3. Fibromatose and General Paralysis. Dr. A. Cullerre.

1. *Women in Religious Orders, etc.*—The author presents an interesting study (to be continued) of the lives and mental and physical experiences of three nuns of the famous convent of Port Royal, since destroyed (near Paris, France). The facts are obtained from various historical, biographical and statistical works cited by the author, and the study carries us back to the year 1661, the time of the Jansenist controversy, when the nuns, upon pain of dismissal to other and, in the Catholic sense, orthodox communities, were required to sign a "formulary" condemning certain five propositions contained in Jansen's treatise upon the theology of St. Augustine. When we consider that the propositions were upon such subjects as Free Will, Divine Grace and Predestination, we cannot wonder that the sisters. who had sought rest for their souls behind the convent veil, should have been driven into torment of mind, when compelled to decide whether they were bound in conscience by the decision of the church on those subjects, while in their own understanding, they held different views. Under stress, some would sign, and thereupon be seized with mental torture, dreading the fate promised to liars and hypocrites. Mind acted on body and aggravated physical maladies. The author, after stating at much length the story of fifteen years of bodily illness and of occasional moral suffering, in the life of the nun Margarite Duprê, and relating particularly an account given of her entire relief, in answer to a special prayer, from all her bodily illness for three months, makes the following summary of her case.

*Character.*—Haughty and of a "nature prompt and lively," she bacame under the influence of religious suggestions, reserved, quiet and humble. She called herself "a poor ignorant" and busied herself with labors to the most lowly.

*Sadness.*—She exhibited a predisposition to sadness. In 1661, dysentery declared itself upon an occasion of mental distress. In 1662 her abscesses of the liver were accompanied "with great spiritual suffering." In 1664, during her exile to the "Anonciades of S t.Denis," the news that certain nuns of Port Royal had signed the formulary plunged her into dejection. Herself urged by the archbishop of Paris to surrender, "she passed five days in weeping, in despair of her condition, distracted between the fear of offending God in signing and having always to suffer for not signing," "in pain and transported with agony." She finished by submitting and gained only new disquietude. The day of her return to Port Royal, she remained in a corner of the church "praying and weeping." At that time "she slept little at night." Finally, being convinced that the signature was a wrong act, she accused herself of her fault "in terms most humble and most touching and with great abundance of tears." "From that moment she felt the greatest joy that she had ever experienced, mingled with heartfelt grief for her sin." She continued to deplore it and had frightful remorse on account of it.

*Suggestibility.*—She said *à propos* of the pseudo miraculous, as the author calls it, remission of her illness: "I have not only received that grace by the intercession of our dear mother, the deceased abbess Mother Angelique, but also some other grace, very secret and very considerable, that I am not able to publish; and I can give assurance that I am never inspired to demand anything of God that I do not obtain it, by her intercession." This gives the measure of her suggestibility. She had made her profession at a convent of the Congregation of Notre-Dame in Flanders, when "at the end of two years the 18 December, 1651, she was received at Port Royal, without *dot*." There she receives the suggestions of Antomé Singlier, director of the nuns and of the abbess Jacqueline Arnaued, who, says she, "takes possession of my heart." Having refused to sign the antijansenist formulary of 1661, she was in 1664 exiled to the Annonciade of Saint-Denis. Here she made nine novines daily. In the first months, she gloried in her resistance, but at the end of ten months of captivity, "she allowed herself to be so carried away by considerations of blind obedience to her superior, that forgetting everything else, she signed the formulary purely and simply under the direction of M. de Pèrefixe," and she did it "with great uneasiness of mind." "She was then ill and feared to die in criminal disobedience." "She signed also the new formulary by the mandate of the archbishop." Returned to Port Royal and again submissive to Jansenist suggestion, she retracted her signature and refused to sign the formulary of 1665. Besides, wishing to multiply the proof of her repentance, she made several copies of her retraction, which she cast from the windows and underneath the walls of the monastery, at that time blockaded. She died at Port Royal in 1666. As to the supposed miraculous answer to prayer, the author considers it either merely a coincidence or that it was produced by "auto-suggestion."

The second observation is upon the case of the nun, Anne-Marie de Flécelles de Brégy, who was a relative of Cardinal de Retz. She had a strength of character which nothing could move. "I am seized," she writes in 1664, when of the age of thirty-one years, "with a certain fear, that I have not enough of fear." And *à propos* of the nuns, who had signed the antijansenist formulary, it has put me in such a trembling and terrible fear lest God should abandon me, that I have instantly supplicated him for grac eto cling to him and to suffer all my life humiliations, afflictions and maladies of every sort to obtain the help of his grace and the gift of perseverance never to abandon the truth." "Why shall I not tremble? Why shall I not fear, when we see those fall, by whom one expected the salvation of Israel—It is a sorrow the most affecting in the world."

She was exiled to the Ursulines of Saint Denis, but returned the next year and signed with her blood a declaration that she persisted in her refusal to sign and disavowed all that she should be made to do in her last illness. She resisted to the end, dying at the age of fifty-one.

The third observation is similar in character. The author's conclusions are to follow in later number.

2. *Treatment of Facial Neuralgia.*—This is the conclusion of a previous contribution, relating to the department of Electrotherapy in the Charcot Clinic. The author finds it difficult to explain that certain monographies no longer insist upon the electrical treatment of facial neuralgia and that others pass it by in entire silence. Does not, he says, Gilles de la Tourette affirm that if medical means fail, there still remains surgical treatment, treatment full of dangers and chances, but outside of which there is nothing to try? Dòes not Mauclaire also say that when medical means have been utilized without success, the physician "*ought to give place to the surgeon,*" and does not this author counsel to operate at once and without waiting, under the pretext that to wait is to give the nevrite ascendency and to the encephalic lesions time to evolve? And he adds: "Although the re-

sult should not be perfect (Trousseau affirms that he has never seen a complete cure of tic douloureaux of the face), we can say with Monod that one year of well being in the course of an affection which imposes veritable torture constitutes a benefit appreciated by the patient, so much more, as the intervention is free from dangers (nevrectomy)." The author, contrary to this opinion, will not admit that after a conscientious trial of medical methods, the physician effaces himself without hesitation before the surgeon. A place, an important place, he says, should be reserved for electric treatment, which, in all cases, we should have interest to attempt before dreaming of a surgical intervention, however light. The author mentions the various surgical methods and gives some statistics of the great danger of a fatal result in gasserectomy, and in view of the benefit to patient shown in some fifteen cases that he reports at length, he states that he thinks that "course to pursue" should be the following:

(1) In cases of neuralgia, with well determined etiology, we should have recourse to their specific treatment. (2) In neuralgias of a mild type, we should commence by resorting at first to the usual medications, free to recur in case of insuccess to electricity, which will bring about almost certainly a complete cure. (3) In neuralgias of a grave type, after one has established the inefficacy of medical treatments, administered in a methodical fashion (among them opiatic treatment) we should submit the patient *to electrization, during a time sufficiently long* to judge of the effects obtained (minimum three months) and we should not decide to employ a surgical intervention, except in case of *absolute insuccess*. In last analysis a peripheric operation might be tried (névrectomy). But it is only *in despair of the case* (en désespoir le cause) and before the formal demand of the patient or the positive threat of suicide, that we should resolve to propose gasserectomy.

3. *Fibromatosis and General Paralysis.*—The author, in presenting the case of a woman afflicted with dermofibromatose, and of whose desperate condition he gives a photograph, asks: Is the affection, known under the name of the Malady of Recklinghausen always congenital? Is it due to an anatomic alteration of the nervous system? It is not yet incontestably established. Still we understand that the authors admit that, in subjects afflicted with this malady, there exists a certain *congenital feebleness* of the nervous system, which places them in a category apart from degenerates. Upon this point, the case we present and which, believe us, offers a case hitherto unique of general paralysis in a woman afflicted with congenital and probably hereditary dermofibromatose, may have a certain interest. The patient was a woman, aged forty-seven, a street vendor, married and with one child; admitted to the hospital in April, 1901; chronic alcoholism, dementia, impulsion to suicide, vague idea of wealth and afflicted with generalized fibromatose of the entire external tegument The back from the neck to the sacrum is literally covered; upon a foundation of little sessile fibromes are attached numerous soft excrescences with large pedicule, particularly in the median region. The patient affirms that "she had that from birth," and that her mother was afflicted with similar tumors. June 1 she was attacked with erysipelas of the face, following an excoriation of the nose, which later was cured. June 6 an epileptiform attack. In December further series of like attacks. January 5, 1902.—Grippal enteritis of grave form, followed with cachexia, slough of sacrum and progressive marasmus. May 6.—After a series of attacks, the patient, profoundly cachectic, succumbed.

*Comments.*—We do not know whether, aside from alcoholism, we can attribute any other cause in this case of general paralysis. We are ignorant, notably, whether this patient has had syphilis. The only thing besides which appears to us interesting in this observation, is the coincidence of fibromatose, an affection congenital and familial, as the patient said (and there is no reason to suspect her veracity, for she has never varied on this point in spite of her state of dementia), and of peri-encephalitis, a malady held as the most accidental of psychopathies, and considered generally as developing ordinarily otherwise than from predisposition and in individuals with brain primitively well constituted. Or, if fibromatose is the index of a *congenital feebleness of the nervous system,* following the exact expression of the authors, this case should be added to those already numerous, where we see general paralysis attack the predisposed, the degenerate, even also imbeciles. In 1888, L. F. Arnaud published two cases of general paralysis in imbeciles, recalled two analogous observations, one of Morel, the other of Christian, and attributed the congenital cerebral feebleness of the patients to the alcoholism of parents. In 1893, my interne, Gagnerot, in his thesis, entitled, "Of Predisposition in General Paralysis," gave some examples of paralysis, grown upon the soil of degeneracy. In 1897, L. Chappelletti published an article upon general paralysis in imbeciles. In 1898, I myself, in the *Annales Medico psycho-logiques,* gave observation of the same kind and advanced the opinion that "almost all general paralytics of the rural class are weaklings." "The observations which I have collected since are not of a nature to make me change my opinion.

RICHARDS (Amityville).

## NEUROLOGISCHES CENTRALBLATT

(Vol. 22, 1903, No. 5, March 1.)

1. The Acromial Reflex. W. v. BECHTEREW.
2. The Carpo-metacarpal Reflex. W. v. BECHTEREW.
3. New Contribution to the Physiology of the Tendon Reflexes. Preliminary Contribution. A. E. STCHERBAK.
4. Vesical Incontinence and Paralytic Manifestations in the Extremities in Focal Softening in the Subcortical Ganglion. A. HOMBERGER.
5. Further Contributions upon the Developmental (Myelogenetic) Regions in the Human Cerebral Cortex. P. FLECHSIG.

1. *Acromial Reflex.*—Bechterew calls attention to a periosteal reflex obtained by striking with the percussion hammer, the acromial portion of the scapula and the coracoid process. This reflex consists ordinarily in a flexion of the forearm, sometimes in an inward rotation of the hand and in exaggerated cases in a flexion of the fingers. These movements are produced by a contraction of the cervico-brachialis and biceps muscles. The reflex is obtained in any condition where there is a heightening of the reflexes as in organic hemiplegias and in amyotrophic lateral sclerosis.

2. *Carpo-metacarpal Reflex.*—Bechterew describes a periostal reflex obtained by striking the carpus and the adjoining metacarpal region resulting in a flexion of the fingers with the exertion of the thumb. The patient's hand should be held with the dorsum upwards. It is obtained in organic lesions situated above the cervical swelling, especially in organic hemiparesis or hemiplegia, as of cortical or capsular origin.

3. *Physiology of Tendon Reflexes.*—Stcherbak records some interesting experiments upon animals. By means of a tuning fork rapid vibrations to the hind leg of rabbits were applied, which resulted in a heightening of the knee reflex, knee clonus, which could be produced by tapping, and passive movement of the knee joint and spastic tremor. These phenomena could be produced by irritation of the other foot. Rapid passive movement 1000 to 1500, also produces a similar condition. Cutting the spinal cord above the center for the knee reflex and applying rapid vibration produced a heightening of the knee reflex, but no clonus or spastic tremor. These vibrations had no influence upon the animal's general condition or muscle

tone. Vibrations applied to the spine, especially the lower dorsal region, produced a spasticity of all the muscles of both hind extremities. Stcherbak concludes as a result of these experiments that by means of vibration we can so influence the lower, somatic portion of the nervous system, that phenomena are produced which we usually ascribe to the higher or psychical apparatus, and that these phenomena may be latent for some time afterwards.

4. *Subcortical Ganglion.*—Homberger describes the symptoms of ten cases in which numerous minute areas of softening were found in the corpus striatum and thalamus. In six of the cases the lesions were bilateral. Incontinence of urine was present in all the cases. Micturition was of the automatic type. There was partial paresis of the lower extremities, and increase of the reflex. The upper extremities were also somewhat affected. Forced laughing and crying and occasional hemichorea and hemiathetosis were present. Homberger concludes the following as the result of lesions in the corpus striatum and optic thalamus: (1) Unilateral lesions cause transient incontinence and a permanent increased desire for urination; (2) bilateral lesions cause permanent incontinence of urine, which does not differ from the spinal form; (3) the subcortical innervation of the bladder is bilateral; (4) superficial lesions do not cause incontinence; (5) bilateral areas of softening in the basal ganglia cause disturbance of station and paralysis which differ from those cases where the lesions involve the cortical fibers of the internal capsule.

5. *The Developmental Areas of the Cortex.*—Flechsig gives the result of his study of six more brains with regard to the date of the appearance of medullary sheaths in the various tracts of fibers proceeding from or to the cortex. This makes a total of 52 brains which he has examined. He modifies slightly his previous statements: (1) Flechsig divides the cortex into 36 regions. He adds a new area to the first parietal convolution, making three areas. This region is found near the intraparietal nucleus and resembles the subangular convolution in size, the arrangement of its fibers and the date of appearance of the myelin sheaths. He is not certain that fibers from the corona radiata enter this region. A new area is distinguished in the upper portion of the occipital convolution, making three areas in the sight region; (2) regarding the order of development the following is of importance. The motor convolutions show the most marked development in the more fully developed fetuses. In a brain of a 34 c.m. long fetus he found a well developed bundle of fibers which were traced to the olfactory sphere, while the fibers of the cerebral convolutions were not differentiated. Unless this is an exceptional case, the olfactory sphere should be designated No. I, and the central convolution as No. II; (3) it is possible to distinguish between the motor and sensory bundles of fibers. The motor fibers are found anterior to the central fissure and the sensory fibers posterior to this. In the supra and subangular convolutions the types are not distinct: (4) in 1901 Flechsig demonstrated that to every sensory (centripetal) bundle there corresponds a motor one. He considers now that the projective system of the brain cortex is composed of similar conjugate pairs of bundles; (5) the order of development of the brain sulci has an important bearing upon the order of development of the myelin sheaths. The areas indicated as primary appear very early while the sulci termed as terminal appear late. This is not definitely proven, but the relation is established.

(Vol. 22, 1903, No. 6, March 16.)

1. Contraction of the Sphincter Iridis in Pupils Immobile to Light, in Accommodation and Convergence Reaction. M. Rothman.
2. Anatomical Changes After Crushing the Roots of the Spinal Cord in Dogs. S. Bickeles.

3. Neurotonic Pupil Reactions. J. PILTZ.

1. *Pupillary Changes.*—Rothman reports a case of a girl twelve and one-half years of age who had occasional periods of unconsciousness during childhood. At five years of age she developed periodical attacks of migraine. After one of these headaches it was noticed that the right pupil was dilated at maximum and was absolutely immobile. The left pupil was normal. Potassium iodide was given. After a period of five months there was a slight reaction of the pupil in accommodation and convergence. During the whole period of observation, three and one-half years, no reaction to light was observed. In the course of time the right pupil regained its normal size, but would remain contracted 30 to 40 seconds after accommodation and convergence. The case represents an isolated paralysis of the right sphincter iridis, the lesion probably being a small hemorrhage in the oculomotor nucleus.

2. *Crushing the Spinal Roots.*—Bickeles crushed the anterior and posterior spinal roots of dogs either with pincers or ligatures for five minutes. The dogs were killed from seven weeks to two and one-half months after operation. Examination showed in the posterior roots a large number of fine regenerated fibers, the number depending upon the length of time elapsing after the operation. Bickeles also found what he believed to be regenerated nerve fibers in the intramedullary portion of the compressed posterior roots. This, however, was not very active, probably due to the increase of neuroglia tissue and a sclerosis. Examination of the posterior roots of dogs who died two to two and one-half weeks after operation showed an absence of centrifugal fibers. In the anterior roots a band of fibers was found which were traced as coming from the posterior roots. Degeneration was always on the same side as the injury.

3. *Neurotonic Pupil Reactions.*—Piltz discusses the inportance of pupillary phenomena. In 37 cases of progressive paralysis, 11 did not respond to light on either side; 10 in whom the light reaction failed in one and was poor in the other; 7 in whom the reaction was poor in both sides; 4 in which only one pupil reacted poorly and 5 in whom the light reaction was normal. In 63 cases of tabes, in 37, both pupils failed to react to light; in 6 the pupils on one side failed, the other being slow; in 15 both pupils reacted slowly; in one, the pupil on one side reacted poorly, the other normally, an din 2 cases the light reactions were normal. Impairment and reaction manifests itself either that the pupil does not contract as much as it should or that it does not contract as rapidly as normally. It also manifests itself in a persistence of the narrowing of the pupil for some time after reaction. Four cases are recorded with the later phenomenon.

WEISENBURG (Philadelphia).

## MISCELLANY

A NOTE ON TRAUMATIC SYRINGOMYELIA. Alfred Gordon (Philadelphia Medical Journal, May 9, 1903).

The writer reports a case of a woman aged forty years who, ten years before, had slipped and fallen on her back. This was followed by numbness and a sensation of pins and needles, and burning and aching in both hands. The pain disappeared but the numbness persisted. The hand would not be pained by burning, though covered by blisters. Three years ago a red swelling on the palm of the hand developed, which increased. A subsequent dislocation of the arm was painless. The motor power in both lower and right upper extremities was normal. The left arm was weaker and larger than the right, each finger being larger than the corresponding finger of the right hand. The subcutaneous tissue seemed hypertrophied. No hysterical stigmata were noted. The skin was soft and thickened. The reflexes and reaction to Farradism were diminished on the left arm.

Anesthesia of side of face and head was present. Temperature sense (heat and cold) were lost over the whole left arm and half the thorax to sternum and the middle line of the spine. Loss of pain sense was identical with thermo-anesthesia. Very little muscular wasting was present. The case was evidently a typical sensory dissociation characteristic of syringomyelia, also trophic changes of the tissue underlying the epidermis. The cause was probably the traumatism causing a hemorrhagic focus, developing into a gliosis or cavity of the spinal cord. Trophic disturbance in syringomyelia of the nature of edematous swelling of paralyzed limbs are not rare. A hard edema due to hyperplastic process in the subcutaneous tissue was described by Marinesco under the name "main succulente." The vasomotor and trophic centers for the bones and integument are situated in the gray matter of the posterior cornua, about the eighth cervical.

NOYES (New York).

DO OUR PRESENT WAYS OF LIVING TEND TO THE INCREASE OF CERTAIN FORMS OF NERVOUS AND MENTAL DISORDER? Charles E. Atwood (New York Medical Journal, June 13, 1903).

Insanity is increasing slightly, especially degenerative types. Neurasthenia is increasing. This increase is found mostly in the immigrant population, 75 per cent of the insane of the State and 80 per cent of the cases of neurasthenia at the Vanderbilt Clinic being either foreign born or of foreign parentage.

Our present methods of living entail an increased mental strain, the number of sensory impressions and variety of ideas forced upon us by our increasing interests permit scarcely time for the reception of impressions, and no time for their proper assimilation. Emotional stresses, e.g., those caused by sudden and great reverses of fortune; profound impressions induced by fanatical doctrines, impressions induced by certain lurid daily newspaper are deleterious to the neurotic. Over indulgence in eating and drinking tends to arterial sclerosis. Tendency to auto-infection is assisted by irregular living, and auto-infection is a recognized cause of certain forms of insanity and certain neuroses. The immigrant suffers the most on account of competition with a superior race, deficient alimentation and resultant enemia, other concomitants of poverty, and faulty hygienic conditions. The consumption of alcohol per capita in this country has increased, and our women are drinking more and more each year. This has a tendency to establish a faulty heredity and induce a strong predisposition to nervous disorders. AUTHOR'S ABSTRACT.

BRACHIAL PARALYSIS—POST NARCOTIC. F. J. Cotton and S. W. Allen (Boston Medical Journal, May 7, 1903).

The writers report four cases of post-anesthetic paralysis, and collect thirty from general literature. The patient is anesthetical with the arms drawn up over the head, and after recovery one or both arms are found paralyzed. There is little or no pain and slight sensory disturbance. The muscles of the shoulder and arms are affected in a varying degree—sometimes all are involved; sometimes the shoulder is spared, and the hand and arm muscles are alone affected. In a few days there is beginning of return of function, more usually in the hand, then a slower return of power in the extensors to the deltoid and to the rotators of the shoulder. As the function returns many of the cases present a paralysis approximately of the "Erb" type (deltoid, biceps, brachialis anticus and supinator longus). In a very few cases there is involvement of the sympathetic root communicating with the first dorsal root, signified by pupillary and other ocular changes. Later, soreness of the nerve trunks and of the muscles may appear. Reflexes are usually normal. Electrical tests rarely show a partial reaction of degeneration; usual reactions are normal. There are three possible mechanisms by which these paralyses may occur: (1) Pressure on the plexus, (a) exerted between the clavicle and transverse processes of the vertebræ, and (b) between the clavicle and the first rib; (2) tension on the roots or plexus from the position of the arm; (3) tension from position of the head or neck, the arm being abducted.

NOYES, New York.

IMPERATIVE CONCEPTIONS. L. Harrison Mettler (Medical Record, April 4, 1903).

Imperative conceptions or ideas are those involuntary, unrelated persistent notions that spring up in the minds of many victims of nervous disease. Though found in many states of mental and physical debility, they belong essentially and entirely to the psychoses. A sharp distinction should be made between the imperative conceptions of the sane and the insane. In the sane the person's judgment remains unaltered; he tries to resist them and often succeeds. The imperative conceptions of the insane become for the time being the patient's very ego. The insane delusion is deeper and more influential than the imperative conception, entering into the constitution of his ego, and coloring and outlining his objective world. The so-called imperative idea, on the other hand, is recognized by the patient to be a mere subjective phenomenon, annoying and persistent though it may be. It may involve all the faculties of the mind, not alone the imagination, but there are usually no distinct hallucinations or delusions with these impulsive conceptions. Insane delusions are, primarily, the result of a diseased cortex; imperative conceptions are the result generally of disease outside the brain.

NOYES (New York).

NEURITIS FROM WHOOPING COUGH. Charles E. Aldrich (N. Y. Medical Journal, June 6, 1903).

Neuritis may attend or follow any of the acute infections, especially those characterized by prolonged high fever, marked disturbance of nutrition and great prostration. The author reports a case of a child aged four, who, after whooping-cough, developed weakness, pain in the legs, weakness of grasp of hands, nasal speech and regurgitation of fluids from the nose, diminution of knee jerks and loss of touch and pain sense in the hands and feet. Recovery came after twelve weeks. Various forms of paralysis have been observed to complicate or follow whooping-cough. Literature proves that it is rare. Sixty-two cases reported were cerebral in origin. Several cases of general peripheral neuritis were collected from literature.

W. B. NOYES, New York.

DEVELOPMENT AND CARE OF CHILDREN. J. C. Cook (Journal of the American Medical Association, June 6, 1903).

The changes in the nervous system from birth to maturity afford us many examples of the differences between the child and adult. At birth the central nervous system, as well as the peripheral, is almost entirely unmedullated. Hence we find the fibers of the spinal cord, the medulla, corpus quadrigeminus and the pons varolii mainly somatic. The motor nerves receive their attachments much earlier than the inhibitory centers are developed, hence the objectless movement that characterizes the unmeasured exercise of the young. Thus maturity is gradual, and full growth is only reached between puberty and adult life. It is our duty to guard and conserve every atom of energy through this period of unfolding and budding life, realizing that they are bundles of possibilities unsolidified, which begin at the beginning and end we know not where. The study of biology teaches that the higher the organism, the slower the development, hence the necessity of a long period of protection and care. Take with this the long periods of stress and functional demands on the nervous system and we can realize the greater necessity for a wise and careful guardianship. The writer urges strict control of the question of child labor, and advises a commission of five members, at least two of whom shall be physicians, to have jurisdiction over all educational and labor questions.

W. B. Noyes, New York.

Effects of the Streptococcus on the Cortical Nerve Cell in Meningitis. Leonard K. Hirshberg (Maryland Medical Journal, July, 1903).

Autopsy: Male, thirty-nine years. Clinical diagnosis: Fractured skull, cerebro-spinal meningitis, myocarditis, endocarditis, pyemia, pneumonia at base of right lung.. Anatomical diagnosis: Purulent traumatic cerebrospinal meningitis, depressed fracture of skull; rupture of dura, depression and bruising of right cortex; fatty liver. Brain: Subarachnoid space contains exudate, consisting of two zones. Inner zone, in contact with cortex, consists of network of fibers, including many cells with long vesicular nuclei, surrounded by much less staining cytoplasm. Probably proliferated endothelial cells from serous surfaces and lymph spaces of membrane. Few poly morphs nuclear leucocytes in the zone. Outer zone consists, almost, of poly morps nuclear leucocytes. Tissues of pia richly infiltrated and thickened by exudate of pus cells, in which numerous proliferated endothelial cells are made out. Weigert's method shows moderate number of streptococci in exudate. Cortical sections, from area of Rolando, stained by Nissl, show disappearance of tigroid bodies from cells. Many cells also exhibit loss of nuclei. Cells shrunken and distorted where nuclei remains. Nuclei swollen, vesicular, limiting membrane at times in contact with body wall of cell. Eccentricity frequent as to nuclei and nucleoli. Direct or immediate action of the toxin of the streptococcus seem probable.

J. E. Clark, New York.

The Epileptic Aura. William P. Spratling (Medical News, July 18, 1903).

Four types of auras are studied: (1) Psychic, (2) sensory, (3) motor, (4) irregular. In a close analysis of 815 males and 510 females, 36 per cent were found to have a sensory aura of some sort, 4 per cent had a psychic aura, and 2 per cent a motor aura. Of the entire number, 45 per cent had some aura, 55 per cent none. The sensory auras are not only common, but extremely varied, affecting any of the special senses, including marked perversions of the same, and being brief or rather prolonged. Visual auras predominate, and appear either as flashes of light or colors, or optical delusions, or temporary blindness. The epigastric aura appears in 15 per cent of all cases, and is the most frequent. The problem in epilepsy is to determine whether the initial disturbance, the thing that breaks up the rythmic periodicity of the respiratory impulse, is to be sought in the respiratory center primarily, or whether it lies somewhere in the periphery, in the stomach, or elsewhere, and represent the original source of the epilepsy. For instance, when a case with epigastric aura suffers from periodic indigestion, gastric catarrh or flatulence, the probability is that the same cause makes the aura and the flatulence, and there is some subtle agent in the unknown field of chemical pathology.

W. B. Noyes, New York.

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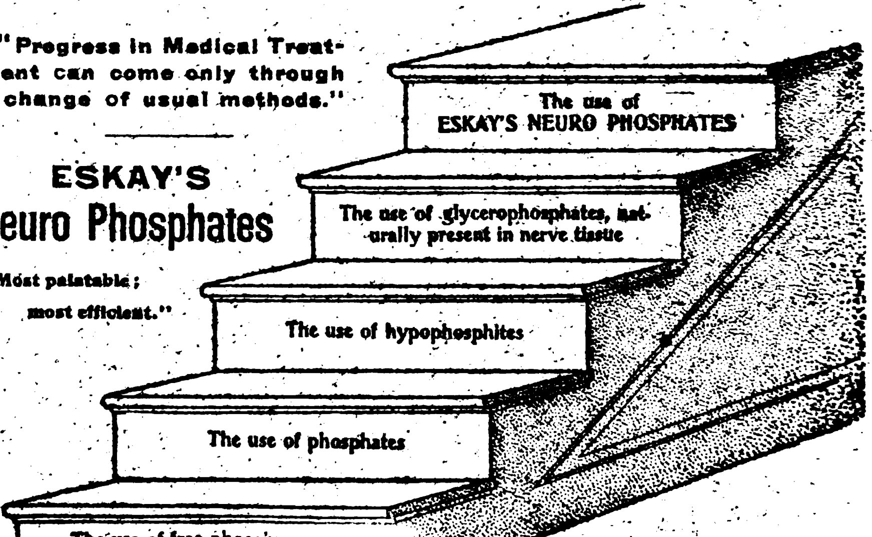
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Vol. 30 November, 1903 No. 11

# The Journal of Nervous and Mental Disease

The Official Organ of

The American Neurological Association

The New York Neurological Society

The Philadelphia Neurological Society and

The Chicago Neurological Society

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Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E. Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

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Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

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SSUED MONTHLY $5.00 PER YEAR

FOREIGN SUBSCRIPTION, $5.60 PER YEAR.

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3,

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Volume 30. November, 1903. No. 11.

THE

Journal

OF

Nervous and Mental Disease

COLL NOV 7 1903 CAMBRIDGE, MASS.

## Original Articles.

### THREE CASES OF TUMOR INVOLVING THE SPINAL CORD; TREATED BY OPERATION.[1]

By Dr. James J. Putnam

and

Dr. J. W. Elliott

These cases are reported in order to show the benefit that may result from operations for tumors involving the spinal cord, even when it is not possible to effect a complete cure; and also to illustrate the danger with which such operations are attended.

In the first case to be described the result was favorable to a degree far exceeding our expectations. It may be that the operation was only partially responsible for the eventual improvement, but it cannot possibly be doubted that it furnished the controlling factor.

Case 1. The patient, who was a young man, twenty-three years old, in the employ of the West End Railroad Company, was brought to Dr. Putnam in December, 1897, and gave the following history:

He said that, just three years before, he was doing a job of work which required him to mount on one of the tall ladders reaching to the trolley wire, and that while descending he missed his hold and fell. He grasped the ladder as he went down and in that way broke his fall, but at the same time his neck received a sudden twist. He picked himself up without help and walked a distance of about a quarter of a mile, suffering only from pain in the neck; and then took a car home.

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[1] Read at the meeting of the American Neurological Association, May 12, 13 and 14, 1903.

That night he suffered from pains in the neck, and for two or three weeks afterward he stayed in the house, not feeling able to work. There was no disturbance of motion or sensation of any kind, except that his head was held somewhat askew, and that attempts to turn it to the left caused pain in the neck.

Six weeks after the accident he went to work again, and worked for another year, feeling well, but not able to turn his head quite freely from side to side. Then he again received some slight injury, in consequence of which the neck once more became actively troublesome for a time.

In December, 1896, that is, a year before the first examination was made, he was off work for two weeks; then he worked again until April, 1897, when he gave up for good and all, on account of renewed pain in the neck.

In the following month, that is, in May, 1897, he fell while running—perhaps because he tripped, perhaps because new symptoms were coming on—and on this occasion also the neck was painfully jarred.

From that time on he was unable to run, though for several months more he could walk, he thinks, nearly as well as ever.

The evidence up to this point makes it clear that the original injury was the primary factor in setting up the subsequent morbid process, and this conclusion seems the more justified from the fact that he was never able to turn his head freely after his first accident, and less well to the left than to the right.

In July, 1897, his gait became awkward, and in August his hands began to feel numb and would shake on use.

For several weeks before the first examination his back and sides had been feeling weak, and breathing had been getting more and more difficult. There had been no paralysis of the sphincters.

The physical examination made on December 27, 1897, showed that the movements of both arms were weak, and that the difficulty of motion increased distally. Thus, his fingers were semi-flexed, and further flexion or extension was impossible; flexion and extension at the elbow were possible, but very feeble; abduction at the shoulder was better but also feeble, and not so good on the right side as on the left. Passive extension-movements of the fingers excited strong clonus.

The prick of a pin was felt fairly well on both hands, but, unlike the motion, it was better for the left side than for the right. [Brown-Séquard complex.] Light contact was felt on both hands and arms, and this form of sensibility seemed to be less affected than that of pain. The movements of the legs were also weak, but were all possible. The right leg was stronger than the left. The knee-jerks were greatly exaggerated, and ankle clonus was pressent. The respiration was labored, and the accessory muscles of inspiration were called strongly into play. The temperature, by the mouth, was 99.7° F. On palpation in the pharynx the bodies of the upper cervical vertebræ seemed to be unusually prominent.

The presence of this group of signs—the involvement of all four limbs, of all the costal muscles, and even of the diaphragm—made it obvious that the patient was suffering from a lesion high in the cervical region, and a lesion of such a character as to affect, in some measure, the whole thickness of the cord, the centripetal as well as the centrifugal tracts. The whole course of the disease, the history of neck-injury resulting perhaps in partial dislocation of the cervical vertebræ, the prominence of the upper cervical bodies in the pharynx, all pointed to an affection of the bony structures, while the high exaggeration of the wrist-jerks and knee-jerks bore out the idea that the nutrition of the cord was being impaired rather by pressure, with its attendant circulation disturbances, than by a destructive lesion primarily involving the neural tissues themselves.

It was thought at first that the bone disease was tuberculous, but Dr. Walton, who made an examination at a later period, favored the diagnosis of a sarcomatous growth at the seat of injury, and this proved to be correct.

The patient was at once referred to the Massachusetts General Hospital, which he entered on December 28, 1897, coming under the care of Dr. F. C. Shattuck. At entrance his temperature was 100.2° F., pulse 112, respiration 30. The thoracic and abdominal organs were normal. The arms were constantly held semi-flexed and the fingers nearly closed, but he was able to walk up and down stairs with the aid of one man.

After staying a few days, the patient was discharged at his own request, but soon found his legs getting weaker and his breathing more labored, though he did not lose weight.

A few months later he was again seen by Dr. Putnam, and operation was advised, at least for exploration. With this in view he re-entered the hospital on June 19. At this period his fingers were immovable, and his arms and legs utterly helpless, though not absolutely paralyzed. Micturition was voluntary, but the movements of the bowels were secured with difficulty. The tendon reflexes were highly exaggerated, as before.

The head was partly extended, and could be moved to about three-quarters of the normal extent, in all directions; a resistance could be felt deep on the left side of the neck, at the level of the second and third cervical vertebræ. A prominence corresponding to the body of the second cervical vertebra was to be felt in the pharynx.

An attack of difficult breathing precipitated the operation, which was made on June 20, the day after his entrance.

*Operation.* The patient was placed face down, with his head bent sharply forward over the end of the operating table. An incision was made from the occipital protuberance down in the median line nearly to the seventh cervical spinous process. The first, second and third vertebræ were exposed and their laminæ removed. The posterior surface of the axis was found to be eroded, so that on grasping it with forceps it came away as a thin shell of disintegrated bone, disclosing a small grayish lobulated tumor, the size of an acorn. This had every appearance of a sarcoma and was removed with scissors and forceps. It seemed to involve the dura and perhaps the cord itself. The base was curetted, and the curette followed the disease around into the body of the second vertebra, care being taken not to injure the vertebral artery. A quantity of the disease was then removed from the vertebra in front of the cord. Horsley's spine forceps were found to be admirable for cutting the laminæ in this very deep region. The operation was difficult and severe.

The wound was closed without drainage, and the patient made a good recovery from the operation. His respiration at once became much easier than before the operation. There was no pain, and the wound healed by first intention. Within three days the patient discovered that he could move his legs better than before, flex and extend his right hand at the wrist, and move his thumb and fingers.

The pathological report on the specimen removed was as follows:

"An irregular mass of tissue with some spiculæ of bone on it. Microscopic examination showed solid masses of irregular round cells interspersed with large multinucleated ones. Giant-cell sarcoma."—Wm. Whitney, Pathologist.

On June 29 he could flex both forearms on arms to a right angle, and could also flex both thighs. All voluntary motions were much increased over what was possible before the operation. Improvement was reported in movements of arms, legs and neck. Respiration was quiet and natural. July 2, slow but steady improvement was reported in movement of arms, legs and neck.

July 11 he was discharged from the hospital, "relieved."

On April 18, 1899, the patient was re-examined at his house and gave the following report:

He left the hospital a good deal relieved, he said. After getting home he at first lost a little, but then improved again so that, with assistance, he could move about the room a little. He could also lie on his side, in bed, which before the operation had been impossible. After this he grew worse once more and was pretty poorly for several months, although he never lost all that he had gained.

In October, that is, three months after leaving the hospital, the improvement recommenced, and from that time on it had continued without interruption.

At the time of this examination he could raise his hands freely, though rather awkwardly, to the head and above the shoulder, could feed himself with the left hand, and could hold a book and turn the leaves. His fingers were stiff and weak, especially the little finger, but all movements of them were possible, whereas before the operation they were wholly incapable of motion. The legs were stiff, but here also all movements were possible, even those of the toes, though the attempt to move them would bring on extension clonus. He was not able to walk wholly alone. He could turn the head freely to the right, and could move it forward and back, but could not turn it much to the left. The breathing gave him but little trouble, and was both costal and diaphragmatic. He had no pain. His flesh and color had improved. The bowels moved freely and the micturition was normal. The knee-jerks were very markedly exaggerated, as well as all the deep reflexes of the four limbs. His sensibility to contact, pricking and changes of temperature was almost, if not quite, perfect over all parts of the body below the head. The sense of position was good. The improvement continued, so that on the following Thanksgiving Day he was able to walk downstairs and take part in the family dinner.

A final examination was made on January 2 of the present year, 1903, with the following results:

The patient reports that he had been steadily gaining, and that he is now keeping a small store entirely alone. His physical condition is as follows:

The gait is very spastic. On turning round he loses his balance easily, and he sways on attempting to stand with eyes closed. There is no ataxia of the arms, and the grasp of both hands is strong. The wrist and elbow jerks are markedly exaggerated, but there is no muscular atrophy of the hands. The knee-jerks are very lively on both sides, and ankle clonus is present. There is no disturbance of sensibility of the hands or face. The pupils are of moderate size and react normally. The pulse and respiration are both normal. All movements of the head are apparently normal, except that of rotation, which seems somewhat restricted. His neck looks short, as if his head had settled somewhat between his shoulders. There is no sign of recurrence of the new growth.

This case is especially interesting in two respects: (1) Although new growths of the cervical region have several times been reported, this is the first instance in which an operation has been made for a tumor at this level, the one which comes nearest to it being that which is described in the recent and interesting report by James W. Putnam, William C. Krauss and Roswell Park,[2] when a sarcoma was successfully removed from the neighborhood of the fourth and fifth cervical segment. (2) It is important as showing how much can be accomplished even where the disease with which the surgeon has to deal is of the degree of malignancy that belongs to the sarcomas; and how long the tendency to recurrence may be delayed. It was equally true of the case described by J. W. Putnam, Krauss, and Park, that the tumor was sarcomatous, and the symptoms were equally serious with those exhibited by our patient, the disorder of sensibility, indeed, even greater; yet here, also, improvement seems to be still going on after an interval of eight months.

Case 2. This patient was a woman, Armenian by birth, married, thirty-two years old. She was first seen by Dr. Putnam in February, 1902, on account of intense pain through the body and thighs, increased by the slightest movement, and for muscular weakness, which, together with the pain, kept her immovably confined to bed.

The preliminary examination seemed to justify the diagnosis of new growth, probably carcinoma, involving the spinal cord, and on this account she was referred to the Massachusetts General Hospital, where she came under the care of Dr. Elliot. The following notes are taken from the surgical records:

It appeared that twenty-one months before her entrance to the hospital she had been operated on in Worcester for a cancer of the breast, the tumor having been developing for nine months previous to operation. About a year after this she was shaken up in a street railway accident, and one week later she began to feel shooting pains in the sacral region. Shortly afterwards the lumbar region and both flanks became involved, then the left leg, and finally the right leg. In the legs the pain mainly followed the course of the sciatic nerves. So far as she knows, the sensibility of the skin and the muscular power were at that time unimpaired. The pain was always worse on the left side than on the right, and when severe it radiated to the front of the abdomen. From this time she grew steadily worse and became eventually unable to walk, so that for four or five months she had been in bed. Every movement increased the pain, and recently it had become so distressing that she could not be moved in the slightest degree without extreme suffering.

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[2] American Journal of the Medical Sciences, Jan., 1903.

The physical examination showed the heart and lungs to be normal, and the abdominal organs also normal. The muscles of the legs were flabby, but not markedly atrophied. There was no edema anywhere, and no tenderness on pressure over the nerve trunks. An examination of the back showed a small knuckle of protruding bone involving the spines of the last dorsal and the two upper lumbar vertebræ.

While at the hospital she had poor nights, sleeping but a few hours at a stretch, and suffered from intense, cramplike pains in the thighs after resting in one position for any length of time. Several one eighth grain doses of morphia were required every night to secure even tolerable comfort.

The original diagnosis having been confirmed, operation was decided upon and was performed as follows:

An incision ten inches long was made over the lower dorsal and upper lumbar region, and the laminæ of the last dorsal and two first lumbar vertebræ were removed. On opening the canal a new growth, of carcinomatous appearance, was found presenting immediately beneath the bone. It surrounded the nerve roots on either side of the cord, and extended into the tissues outside the vertebræ. This was removed so far as possible by excision and curetting, and two nerve roots on each side, which were at the level of the growth, were divided, inside the spinal canal. The dura was not opened.

In the afternoon the patient suffered from shock, but revived under the use of intravenous infusions and strychnine. During the next few days she gained strength and was fairly comfortable when at rest, though suffering greatly when moved. Her condition varied greatly, and on some days she suffered continuously. The wound healed by first intention. Three weeks later she was reported as looking well and suffering very little pain, but with considerable paresthesia in the limbs. One week afterward, that is, on March 22, she could move the legs more freely, without pain, and suffered but little pain on being moved by the nurse. The improvement in these respects was so great that on March 26, when she slipped out of her chair one day by accident, very little pain was excited. On April 4, that is, nearly a month after the operation, she was reported as feeling well except for slight dragging sensations in the right thigh. On April 7 she was discharged much relieved, and it was felt that the purpose for which the operation had been made, namely, the relief of pain, had been accomplished so far as could possibly have been expected.

The plan of dividing posterior nerve roots had been formed before the operation was begun, and it was even felt that, if no other course offered itself, it might be justifiable to divide the cord above the new growth, so imperative was the need of relieving the extreme suffering.

On leaving the hospital it was found possible to transport her to her home in Worcester, and here she remained living in comparative comfort, still suffering much pain, but not of the same severity as before the operation.

In June a convulsion occurred, followed soon afterward by a second. This seemed to indicate the development of malignant growth in the brain, and within a few weeks from the appearance of these symptoms the patient died.

The best indication of the value of the operation is afforded by the recognition on the part of the patient and her husband that the excessive severity of the pain had been considerably tempered. Indeed, the testimony increases that we should not wait for the assurance of absolute success in the case of either cerebral or spinal tumors. The notable improvement recorded by Dr. J. J. Thomas,[8] from operation on a myeloma of the spine with compression of the cord, is another illustration of the justice of this view.

Case 3. This patient was a man of fifty-four, married, of good previous health, who entered the Massachusetts General Hospital in the service of Dr. F. C. Shattuck, on January 5, 1902.

The hospital records state that five years before, while suffering under the strain of excessive work, he began drinking champagne, and then whiskey, and he finally took up to the amount of half a pint or a pint a day. Three months previous to his entrance he stopped this habit absolutely. He had never had specific disease. Seven years before entrance he had an attack of what was called rheumatism, in the muscles of both sides of the trunk, and was then in bed eight or nine days, suffering intense pain. Since then he had had occasional attacks of a similar sort, but less severe. The pain was sharp and lancinating in character, but did not entirely encircle the body. Two years before entrance he awoke one morning with queer feelings in his head and left half of his body. There was no loss of power, but a sense of numbness and more or less impairment of cutaneous sensibility, accompanied by dizziness. After a few moments he seemed perfectly well again, except that the dizziness persisted for some time. Six weeks later, while at work in his office, he again became dizzy and faint, and this time he lost power in his left side and had a recurrence of numbness, together with tingling sensations. These signs were most marked in the arm. After a few hours his muscular power began to return, although the left arm never fully regained its strength and afterward remained subject to numbness and tingling. Six months before his entrance he began to suffer from pains of intense, radiating character, beginning in the back of the neck and extending through the left shoulder and arm. In the morning on waking he often used to find his head drawn to the left side. The muscles of the hands gradually became weak and atrophic, so that the fingers could not be used for any delicate work; also considerable cutaneous hyperesthesia developed. Two or three months later there was some involvement of the other arm. In spite of the intense pain, he attended to his business regularly. About two months before entrance he began to suffer from a painful girdling sensation from the level of the ensiform cartilage to the groin.

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[8] Boston Medical and Surgical Journal, 1901, Vol. 145, p. 367.

There was no pain in the legs, but they would often shake violently. There was no disturbance of micturition, nor any loss of control over the rectal sphincter. These symptoms were soon folllowed by a loss of power of movement of the legs, which lasted for three weeks, though it finally passed away to a great extent. The muscles of the legs grew wasted at this period in some degree. No mental symptoms occured at any time, nor any headache or vomiting. At times the voice was noticed to be a little husky.

An examination of the blood showed Hgb. 95 per cent; leucocytes 9800.

The physical examination at the time of his entrance into the hospital showed his thoracic and abdominal organs to be negative, and his general condition of nutrition normal. The wrist reflexes were also normal, but the knee-jerks much increased. The Babinski reflex was present on both sides, but most marked on the right. The cremasteric reflex was normal. There was no disturbance of sensibility of the skin of the head and neck. All the muscles of the hands and arms were more or less atrophied, more especially those of the left arm. This atrophy was most marked in the interossei. No hyperesthesia could be made out at any level, but slight stimulations would frequently excite spasms of pain across the shoulders. The grip was weak on both sides, more so on the left. The breathing was almost exclusively diaphragmatic.

An examination of the cutaneous sensibilities of the chest and abdomen showed an interesting dissociation of the sensory functions of the skin. Thus, from the level of the nipple in front and spine of the scapula to the crest of the ilium, behind, and to just above the pubes, in front, there was an almost entire loss of the sensibility to heat and cold, pain, space, and pressure, while the sense of contact in this area was nearly normal. The cutaneous sensibility of the legs was abolished.

The muscles of the legs were almost completely paralyzed, and toe-drop was marked, but atrophy was not present to any extent. Reflex spasms were frequently induced by slight stimulation of the feet.

Massage gave great relief, and the patient used to rest quietly after it.

On January 7th, the patient was examined by Dr. Putnam and Dr. Taylor (also afterward by Dr. Walton), who reported that the signs indicated disease in the lower cervical region, which was primarily external to the cord; that is, either a cervical pachymeningitis or a new growth. It was also thought to be conceivable that the case might be an unusual one of syringomyelia. It was believed that hemiplegic symptoms pointed probably to an independent vascular disease of the brain, unless possibly both might have been due to multiple sarcomata.

Operation was recommended, though the outlook was not considered as promising. It was believed that the lesion must extend high enough to affect the nerves of the fifth or sixth cervical segment, and that it was most severe at the level of the seventh segment. The fact that so much pain was present and was increased by motion, made it probable that the lesion was meningeal, at least in part. The prognosis without operation was considered poor, especially in view of the involving of respiration.

Potassium iodide having been given in large doses without relief and, moreover, being constantly required to relieve the pain, the patient was transferred on February 4th, to the East Surgical Service, and was operated upon, on February 7th, by Dr. Elliot, as follows:

An incision ten inches long was made from the base of the head to between the shoulder blades, and the laminæ of the first dorsal and the four lower cervical vertebræ were removed. As soon as this was done, a new-growth, of grayish translucent appearance, was found encircling the cord and dura like a collar. It occupied the posterior and left lateral part of the extradural space, and was adherent to the dura, though easily stripped from it. This growth had extended longitudinally for a distance of three or four inches, and had spread into the various openings between the vertebræ. A long and careful dissection was made, for the purpose of removing all visible disease. The dura was not opened except at one point, where the growth was found adherent, but from this opening a large amount of cerebrospinal fluid escaped.

The operation was a severe one, and at times the respiration was very poor—partly, perhaps, on account of the patient's position. He reacted well, however, to stimulation afterwards, and slept quietly in the afternoon with the aid of morphine.

The next day he was found to be in great pain, with a temperature of 106 F. and with very rapid breathing. On the second day, February 9th, the temperature had risen to 108 F. The breathing was still more labored, and the patient was semi-conscious. His pulse gradually became weaker, and he died about 2 P. M.

In spite of this unfavorable result, Dr. Elliot felt that the position of the growth and the fact that it was possible to remove it almost wholly without any serious injury to the spinal cord made the operation appear thoroughly justifiable, and that in another similar case the good result which was anticipated might readily follow.

The report of the pathologist, Dr. J. H. Wright, was as follows:

"East Surgical. Stevens. Service of Dr. Elliot. Feb. 7, 1902. Laminectomy.

"The material received for examination from the operation consists of several pieces of grayish translucent homogeneous fairly firm tissue and the laminæ or parts of laminæ of three vertebræ.

"This tissue is intimately adherent to parts of the posterior ligaments belonging to these laminæ.

Microscopical examination of sections from the tissue show that it is composed of small cells and coarse fibered connective tissue. The cells have little or no cytoplasm and seem to consist of nothing more than nuclei which are generally rounded in outline and generally about the size of lymphoid cells. The nuclei are also present. In some places the cells are scattered among the connective tissue fibers. In other places there is little connective tissue and the tissue seems to be made up largely of the small cells. Some capillary blood vessels are present.

"Diagnosis: Fibrosarcoma, probably originating in the posterior ligaments of vertebræ.

"J. H. Wright."

This case thoroughly recalls that reported by Henschen,[4] of Upsala, and Lennander, by whom the operation was performed.

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[4] Cited at considerable length in the Neurological Jahresbericht of Mendel and Jacobson; for 1901. 5th Jahresbericht, p. 507.

Here, also, a similar congeries of symptoms to those which our case presented was held to point to a growth between the 5th cervical and the 1st dorsal segment, and in fact a spindle-shaped, flat tumor was found. It proved to be non-malignant in character and complete recovery followed its removal. It cannot be doubted that our patient would at least have improved greatly, had the operation not been followed by the rapidly fatal symptoms of the first days. The cause of these symptoms is not clear, though this occurrence after opening of the spinal canal is unfortunately, not unfamiliar.

Finally, this occasion may be taken for reporting that the patient from whose spinal canal a fibroma was removed by Dr. J. C. Warren in 1898,[6] has continued to improve steadily and is now comparatively free from annoying symptoms.

A summary of the published accounts of operation for tumors of the spinal region has been so recently given in the paper by J. W. Putnam, Krauss and Park, that it is unnecessary to give further details in this place.

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[6] Report by Drs. J. J. Putnam and J. C. Warren; Amer. Journ. of Med. Sciences. Oct. 1899.

# A METHOD FOR THE RELIEF OF PAIN IN TUMORS OF THE BRAIN.[1]

By William Browning, M.D.,
of Brooklyn.

The hopeless character of most cases of this kind and the limited extent of our resources in treating them are generally recognized. A few cases can be approached surgically (with a view either to the radical removal of the growth, or occasionally to relief by drainage, or to the symptomatic relief of pressure that a trephine-opening affords). A small number respond somewhat to thyroid, a few remain latent, more are amenable to specific remedies, and many are temporarily benefited to an appreciable extent by iodide. The X-rays have been found in a few cases to temporarily numb the pain. These methods are more likely to prove palliative than radical. This brief summary about covers our scanty therapeutics, and even so makes a better showing than many may suppose possible. Granting all these exceptions, however, a majority of cases remains, and in these we are at a loss to know how to meet the conditions. In default of anything better, we grope around for means of symptomatic relief, and usually end by making continuous use of opiates in some form. The latter agents, however, are objectionable for several reasons; they obtund consciousness more or less, and in the long run aggravate pain by increasing constipation.

So long consequently as in the majority of cases of intracranial neoplasm we are unable to apply radical or curative measures, so long shall we need symptomatic agents. This is particularly true of the frequent and most distressing symptom, pain in or about the head. It is the sufferer's chief complaint, and it is especially for its relief that the present plan aims.

While the principal merit of the method to be suggested[2] is that of relieving symptoms, this is done in a less objectionable manner both from the patient's standpoint and from our own than is afforded by narcotics. Nor does it in any way interfere with the use of these if such becomes necessary.

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[1] Paper read before the Brooklyn Society for Neurology, Dec. 18, 1902.

[2] In the discussion of a paper at the 1901 meeting of the American Neurological Association, I briefly mentioned this means of relative relief in brain tumors. Attention had previously been called to the value of vasomotor depressants and sedatives in the headache of syphilis (Brooklyn Med. Jour., 1889, 675), especially in the later forms, and that may have dated in part back to Seguin's use of aconitia and gelsemium for trigeminal neuralgia as early as 1879.

One unquestionable advantage of the use, which, as you know, I have so strongly advocated for a number of years, of the vascular agents (whether to increase or depress the blood-pressure), is that they act in an intelligible way and not in some presumed or speculative manner. Their influence can be pretty definitely foretold and its effect controlled; hence the remedy can be fitted to the case. In other words, they are rational remedies and meet the demand for both honesty and exactness in practice.

The method in the present instance is very simple. It consists in the administration of such depressants as aconitia, veratrum[3] or gelsemium[4] in doses sufficient to soften and control the pulse. The amounts required are not far from those which were recommended in the abortive treatment of apoplexy (*N. Y. Medical Journal*, 1902, Feb. 15). The remedy can be continued as required, though the condition of the circulation should be frequently observed and the amount of the drug regulated according to the necessity of the case and the way it is borne. The dosage in any particular case is kept where it does not dangerously reduce the pulse on the one hand, and yet controls the pain on the other.

This plan may not at any and every instant completely stop pain, but it holds it within bearable limits, and may be all that is required even when the course of the trouble extends over many months. It appears to accomplish quite as much as the method of symptomatic trephining, which was proposed for a similar purpose, and to do this without any of the operative drawbacks.

Doubtless the thoroughness with which pain can be controlled in any given case will depend somewhat upon the manner in which it is produced. Clinically we have, besides those relatively fortunate cases without pain, certain types, and we can trace the source of the pain to at least three factors: (1) There is the local and more or less general increase of intracranial pressure, as occurs most typically in intrasubstantial growths. Where the pain is thus directly due to pressure, it is less intense, but may be more continuous, like a deep ache. In this form marked relief may be expected. (2) Then there is the paroxysmal augmentation of arterial pressure in certain cases, to which reference will be made more fully. It is in these attacks doubtless that the terrific pain-seizures occur, and correspondingly this form should be relieved most of all by depressants. It is really here that we most need analgesics, and that opiates fail except to stupefy. (3) But when on the contrary the pain is the result of direct implication or stretching of external structures, as the meninges or outgoing nerves, and we get instead neuralgias and localized pains, then the good to be realized from depressants may be only proportionate to the degree in which pressure is a factor. And in fact it is supposed that in this last type of cases pain is especially likely to be felt.

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[3] It has been suggested that, as veratrum has a tendency to produce nausea, it is undesirable for this purpose, unless it is the only agent of the kind at hand.

[4] My attention has been called to experimental work indicating that gelsemium "exerts no influence on blood-pressure." Clinically, however, it does affect the pulse, when given in full doses. A better effect might follow its combination with one of the others. As aconite can be given hypodermically it is as yet the best agent for our purpose.

It is fair to ask whether this plan has any injurious effect on the course of the trouble itself. Nothing of the sort has been apparent in any case, nor is there any theoretical reason for suspecting harm. So far as one can decide, life has if anything been prolonged. On paralysis if present it can hardly have much effect either way. Other, in part paroxysmal symptoms, as vomiting, dizziness, spasms, are as a whole favorably influenced. The growth of the neoplasm itself is not visibly affected, certainly not accelerated. Consciousness and the individual's personal control of himself is much better preserved. Moreover, remedies of this kind exert a very favorable influence on a certain tendency in these cases to brain-hemorrhage. This complication is specially frequent in and about the growth itself. Though such hemorrhages are more apt to be numerous than individually large, yet they decidedly aggravate the symptoms of the tumor, and anything tending to prevent their occurrence will be of advantage. Hence, aside from the pain, these remedies act on the other phases and manifestations of brain-tumor favorably if at all.

In occasional cases the depressive agents may also have some diagnostic or differential value.

The points that first suggested a trial of these agents were the increased intracranial pressure, and the pulsatory character often of the pain. In such cases, where warranted by a strong pulse, it seemed proper to seek relief by depressants. Gradually it became evident that in most cases their use practically did away with the need for morphine. Recently a more striking basis for explaining the good effects in some conditions has been afforded by the work of Dr. Harvey Cushing[5].

Perhaps his conclusions, so far as they bear on the present question, can be indicated by a few quotations. Speaking "of the alterations in the cerebral circulation as they are influenced by varying degrees of compression," he says: "When, however, the local process is in the near proximity of, or, if remote, when its effects are so far reaching that the vital centers of the bulb are compromised, the one symptom which with regularity is called forth, and which betokens a serious alteration in the local circulation, is a persisting rise in blood-pressure, which may or may not be associated with a pronounced vagus pulse, with respiratory changes, etc. The length of time experimentally during which these reactions can endure is considerable, even under the extreme degree of elevation—over 200 mm. of mercury from a level of 90 or 100 mm., in many cases—to which we have pushed the blood-pressure." This increased force of the current he figures as an effort of nature to overcome an obstruction. Yet the occurrence of this phenomenon presupposes an already existing increase of the intracranial pressure in the posterior fossæ—a condition that is frequently produced by tumors. The same paroxysmal or longer elevation of the blood-pressure may also have to do with the frequent occurrence of brain hemorrhages in these cases. And yet in the case of a brain tumor it does not appear that any particular good is accomplished by this heightening of pressure. Certainly it must augment any ache. It affords therefore an added indication for the use of depressants in easing and preventing the suffering of these patients.

It is hardly necessary to give details of cases; and the still inevitable ending makes their recital less inviting. If the argument appears sound, you can readily make use of the idea in suitable cases and arrive at your own conclusions. The claim that I present is simply that this in an improved palliative in a most distressing class of troubles.

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[5] Amer. Journ. of the Med. Sciences, Sept., 1902.

# TRICEPS, BICEPS, AND FINGER CLONUS.

By Theodore H. Weisenburg, M.D.,

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Theoretically, there is no reason why clonus of the triceps and biceps muscles should not be obtained when the tonicity of these muscles is increased, but such phenomena probably have not been described. The different text-books on nervous diseases (Gowers, Oppenheim, Mills, Strümpell), and Sternberg's[1] work on the tendon reflexes make no mention of the phenomena.

Triceps clonus was first obtained by me in the following case:

J. N., aged fifty-seven years, was admitted to the Philadelphia Hospital, April 1, 1903. At present he is in the insane department. Diagnosis: General paresis. History of syphilis and alcoholism was obtained. The symptoms began two and a half years ago. He has ideas of grandeur, hears voices, stumbles over syllables. He has some tremor of the lips, and Argyll-Robertson pupils, and the left pupil is larger than the right. The tendon reflexes are markedly exaggerated, and ankle and patellar clonus are obtained on both sides. He has no sensory changes, and no bladder nor rectal disturbances. Tapping the right triceps tendon above its insertion into the olecranon, produces a distinct clonus of that muscle, which is easily exhausted.

Triceps clonus was obtained more typically in a second case, and is fully described in the following report:

W. K., aged sixty-six years, was admitted to the service of Dr. William G. Spiller, in the Philadelphia Hospital, July 30, 1903. His father died with hemiplegia. The patient had not contracted syphilis, but was alcoholic. Four years ago he had a right-sided hemiplegia with aphasia. At present he drags the right leg when walking. The lower distribution of the right seventh nerve is paralyzed. The right upper limb is contractured at the elbow and wrist, and the fingers are flexed. The right knee is also contractured, but all these contractures can be overcome on forced passive movement. The tendon reflexes are very much exaggerated on both sides, more so on the right side. He has ankle clonus, but no patellar clonus. Babinski's sign is present. There is complete right-sided hemianesthesia for all forms of sensation. Incomplete hemianopsia is present. Atrophy is present in both sides, and is more marked on the paralyzed side.

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[1] Sternberg, M. Leipzig ü. Wien., 1893. "Die Sehnenreflexe und ihre Bedeutung."

The patient holds his paralyzed arm close to the chest, the forearm being flexed at a right angle to the arm, the hand being supported by the sound limb. On tapping the triceps tendon above its insertion into the olecranon a distinct clonus of the triceps muscle is produced, lasting at times three to four seconds. It is hard to count the vibrations with the eye or hand, but the number varies between twelve and twenty. Excitement increases the clonic movement, and at these times tapping even with the finger over the body of the muscle almost as high as its upper insertion, in fact a slight jar over any portion of the forearm or arm, will bring on the clonus. Indeed, the patient says that at times, while resting quietly, clonus of the triceps muscle will be produced independently of any stimulation.

The clonus consists in rapid extension and flexion of the forearm upon the arm, the triceps muscle stands out prominently, and in the rapid up and down movement of this muscle its tendinous insertions can easily be made out.

By striking the biceps tendon near the elbow joint with a percussion hammer, there is produced at times a clonus of the biceps muscle, causing flexion and extension of the forearm upon the arm. This is not nearly as constant as the triceps clonus, and can only be brought out on excitement. The resulting clonic vibrations are comparatively slow, there being from five to seven in two to four seconds.

Sometimes percussion over the biceps or triceps tendons will bring both clonus of the triceps and biceps muscles, the triceps clonus outlasting the latter. Again, striking the triceps tendon will at times bring on a clonus of the wrist besides the triceps clonus.

Clonus of the fingers is rarely found. Gowers[2] says that in an increased excitable state of the flexors of the fingers, a contraction, and sometimes by maintaining the increased tension, a clonus can be obtained. Sternberg merely mentions the possibility of finger clonus. No other mention of such a condition was found in the text-books referred to.

W. M., aged fifty-seven years, was admitted May 30, 1903, to the service of Dr. C. S. Potts, in the Philadelphia Hospital. He had right-sided hemiplegia, which developed twenty-six years previously. The man is chair-fast. The right upper limb is contractured at the elbow and wrist, and the thumb is turned inward, and the fingers are clenched. The right leg is contractured at the knee. All the tendon reflexes are increased. There are no sensory changes. Atrophy is more marked in the paralyzed side. Wrist clonus is not obtained on suddenly extending the tightly clenched fingers, the hand being held, but there is resulting clonic vibrations of the fingers upon the carpometacarpal joint. The movements are rapid, consisting in a flexion and extension, are easily exhausted, but soon return, the number of vibrations never being more than from five to seven, and lasting from one to one and a half seconds. Sudden extension of one finger or two fingers will not produce the clonus, but sudden extension of three or of all the fingers will.

I am indebted to my chief, Dr. William G. Spiller, for the privilege of reporting two of these cases, and to Dr. Potts for the privilege of reporting the third.

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[2] Gowers, Sir W. R., 3d ed., p. 23, "Diseases of the Nervous System," vol. I.

# STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL HUMAN BRAIN AND IN DEMENTIA PARALYTICA.

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(*Continued from page* 648.)

Several neuroglia cells as at N, rounded or oval in shape, are to be seen here also interspersed among the nerve cells, and they increase in number proportionate to the nerve cell decrease as the white medullary center is reached. Several capillary blood vessels are to be seen in this layer, cut in different planes. Their walls are made up of a single layer of thin nucleated cells and the vessels filled with red blood corpuscles. The cells of this layer belong to the somatochrome class and either the archyochrome or gryochrome group, depending on whether one regards the chromatic substance as making up a distinct network as in the archyochrome group or as small granules arranged in threads or heaps as in the gryochrome group. It is difficult to determine a network of any distinctness in the majority of these cells, and they seem to the writer to belong more to the gryochrome group. The entire cortex thus measures in this section approximately 2.85 mm. in thickness as it is really an arbitrary division to state just where the gray cortex ends and the subcortical white medullary substance begins, there being a gradual transition from the one to other here as well as between the layers of the cortex. In order to obtain the approximate number and ratio of the nerve cells to the neuroglia cells an effort was made to count the same in various sections of the photomicrograph, counting the nerve cells first and marking each one as counted by black ink, and afterwards doing the same for the neuroglia cells. Upon subsequently using a Zeiss ocular micrometer divided into millimeter squares in a No. 3 ocular with a No. 6 objective, thus giving a magnification of 390 diameters, it was found that the former method, in which only a magnification of 100 diameters was used did not show all the structures present, and those shown were not sufficiently distinct at times to determine accurately their real nature, whether neuroglia cells, portions of nerve cells, etc. With the above magnification,—390 diameters,—and upon focussing in various planes, it was found for instance that a single mass upon the photomicrograph could be resolved into two or more neuroglia cells close to one another and in slightly different planes. By using the above combination it was found that one side of the millimeter square in the eye-piece corresponded to .033 mm. of the stage micrometer and thus of the surface of the section under observation, so that a square millimeter of the ocular network was therefore equivalent to .03323, or .00999 square millimeters (practically .01 square millimeters, which latter decimal was used for convenience in this work) of surface of the section under examination. The ocular micrometer was divided into 36 squares and the number of nerve cells in each of these squares was counted and recorded in seven different fields, in this case in different parts of the section, thus making 252 squares in all. The result was added up and divided by 252 in order to give the average number of nerve cells for each square. This number was equivalent to that contained in .01 sq. mm. of actual surface of the section, or practically 1-100 of a square mm. The total number of neuroglia cells in 108 squares from three different fields was determined in the same manner, the resulting total being divided by 108 to give the average number of neuroglia cells for each sq. mm. of the ocular net-micrometer or .01 sq. mm. of actual surface of the section. These results multiplied by 100 thus will give the number of nerve and neuroglia cells for each square millimeter of actual surface of the section. With this method the average number of nerve cells to each sq. mm. of the ocular net-micrometer was found to be 1.1685; multiplied by 100 gives 116.85 as the average number of nerve cells to be found in each sq. mm. of this section in the second and third layers. In the region just below the middle of the second layer previously described as made up almost exclusively of small pyramidal cells these latter are found very densely packed together, in many parts as many as 2.7 cells were found to each square on an average. This result multiplied by 100 as before gives the high average of 270 nerve cells to each square millimeter in this narrow region, whereas the general average for the entire second and third layer, not including this region, is but 93 nerve cells to the sq. mm., thus showing the great variation in regions of the same section closely approximating one another. The nerve cells of the first layer were so few and scattered that they were not included in this count. In the same way the average number of neuroglia cells to the square millimeter of surface of the section was found to be 125. Counts were also made from a section from the first frontal convolution, somewhat anterior to that from which Plate II, Fig. 3, was taken. This latter block was hardened in alcohol, the section was 10 microns in thickness, and similarly stained with methylene blue. Decolorization was not carried on to such an extent, so that the neuroglia cells were not decolorized, and the section being 10 microns in thickness in comparison to 6 2-3 microns in Plate II, Fig. 3, the neuroglia cells were found to be more numerous. This would seem to indicate that fixing in Van Gehuchten's fluid and staining the same length of time in warm methylene blue results in a somewhat greater decolorization of the neuroglia cells in the following differentiation than in sections hardened in alcohol and treated similarly in respect to staining and differentiation. As stated this section was 10 microns in thickness, whereas that of Plate II, Fig. 3 is but 6 2-3 microns in thickness; but it will be seen by the following figures that whereas there is only a slightly greater number of nerve cells in the former as compared to the latter the neuroglia cells are considerably in excess in the former. Here counts were made in seven different fields of 36 squares each in various parts of the second and third layers for the nerve cells, and in all three layers for the neuroglia cells. The average number of nerve cells was found to be 1.27 to each sq. mm. of the ocular net-micrometer, which multiplied by 100 gives 127 as the average number of nerve cells to the sq. mm. of surface of the section. In the same way the average number of nuroglia cells was found to be 1.64 to the square millimeter of the ocular net-micrometer, which again multiplied by 100 gives 164, as the average number of neuroglia cells to each sq. mm. of surface of the section. The difference in the number of nerve cells in the two sections is not very great,—about 10 per cent, but in the number of neuroglia cells it is much more marked,—over 30 per cent, due partly to the difference in thickness and partly to the greater decolorization of the first section described (Table II). Hammarberg (1895) for counting nerve cells, and Popoff (1894) for determining the ratio of increase in neuroglia cells in acute and chronic cases of Asiatic cholera, used the ocular net-micrometer in a manner similar to that made use of in this article, but in each case in a different line of investigation; the former in the study of idiocy, and the latter, as already stated, in Asiatic cholera. The number of nerve cells and of neuroglia cells, as well as their ratio to one another, varies in the different layers of the cortex. In the first layer there are but few nerve cells, whereas the nuroglia cells are about the same or even more in number than in the other layers, thus making a high ratio. In the second and third layers, however, the cells are the important elements and occupy the greatest amount of space, and although fewer numerically than the neuloglia cells the latter are much smaller, less conspicuous, and the ratio is not so great. Then again the cells are not evenly distributed, but have a tendency to be arranged more or less in irregular groups, so that in some places they are relatively widely scattered, whereas in others they are more closely aggregated, so that a number of counts in various parts of the field are necessary to get an approximate average of the number of cells. The thickness of the section, degree of staining, and extent of differentiation or decolorization must also be considered in Nissl work, as too great decolorization will prevent some cells from being recorded in a photomicrograph, and an extremely thin section would not contain as many cells as a thicker one. Sections 6 2-3 and 10 microns in thickness (two and three clicks of the Minot microtome) were used in this work, and when well stained and decolorization is not carried too far, will show all cells to be found in such a plane.

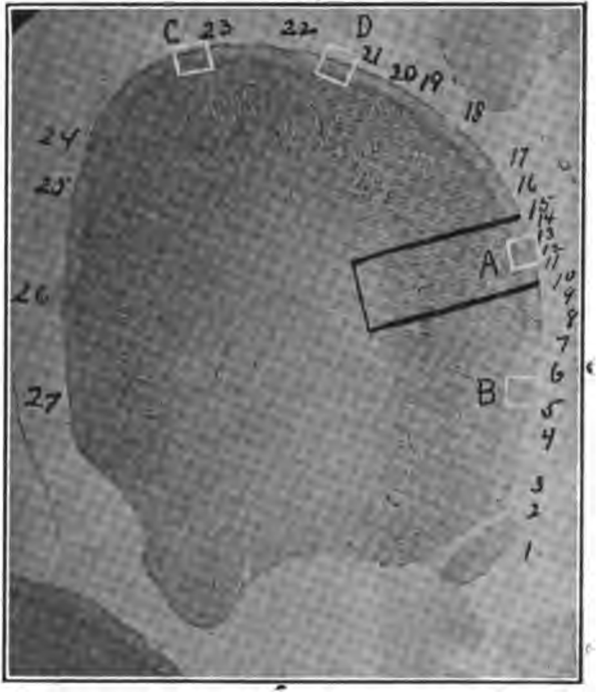


Plate III, Fig. 7.

*Central Region* Turning now to the region posterior to this we come to the motor region of the cortex, comprising the anterior and posterior central convolutions. This region is of especial interest in many ways, and has probably been more extensively studied than any other of the various regions of the cortex. Plate III, Fig. 7, is a photomicrograph magnified 14 diameters, taken from a section 10 microns in thickness at the upper third of the anterior central convolution of Brain A. (Plate I, Fig. 1, 2.) This block was also fixed in Van Gehuchten's fluid, imbedded in paraffin and sectioned serially by a Minot microtome. This section was then stained with warm methylene violet, differentiated in alcohols, 30, 50, 70 and 95 per cent., and absolute, cleared in xylol and mounted in xylol-damar. The actual size of the section which includes the entire width of the gyrus, is .7 of a c. m. in width by .7 c. m. in depth, and taken from a block .4 cm. in thickness, the plane of the section being at right angles to the direction of the gyrus. At the point *a* and outlined in ink is the strip seen in Plate III, Fig. 8, at a magnification of 100 diameters. The outer layer of the cortex is seen to be fairly uniform in thickness, with the exception of the regions indicated at *b* and *c*. At *b* this layer is considerably thicker than in any other part, gradually diminishing in thickness near *a* where it is of the average thickness. At the point *c* is seen a slight thinning of the cortex, which in Plate IV, Fig. 9, results in the entire disappearance of this layer and is intermediate in degree in Plate IV, Fig. 11. This condition will be more fully discussed in the description of these latter plates, and it will be simply stated now that this is an artefact probably due to manipulation. The general radiating appearance of the cortex from the white medullary center to the periphery is well seen here. In the region of the lower part of the second or middle cortical layer even with this low magnification can be distinctly seen the giant pyramidal, or Betz cells arranged singly or in groups. Below this the cortex becomes paler and finally merges into the central medullated white substance. This magnification is especially good for studying the arrangement of the Betz cells. Beginning with the first group in the lowest part of the section on the right, which is the posterior surface of the gyrus, opposite 1 is a single Betz cell of moderate size; just above this, opposite 2 is a group of two Betz cells, one nearer the surface and above the other in the lower part of the second or pyramidal cell layer. Somewhat higher up, opposite 3, and somewhat deeper, are seen two very large Betz cells, one deeper and further from the surface, as well as higher up than the other. Opposite 4 are three Betz cells too far separated to be considered as a single group, so must be regarded as solitary cells. Just above them at 5 are three smaller cells, one above the other at about the same depth of the cortex. Opposite 6 are two small Betz cells, one nearer the surface than the other. Then for a considerable distance no Betz cells are to be seen until we reach a point opposite 7 where a fair sized solitary cell is made out. Just above this opposite 8 are two rather small Betz cells, and a short distance above at 9 a group of three containing two large and one small Betz cell. Opposite 10, and just below the lower ink mark are two very large Betz cells, one overlapping the other, giving the appearance of one in the plate. Just above the ink line opposite 11 is to be seen a group of four Betz cells, one large, and the other three smaller. These and those above and included between the two parallel ink lines can be seen at a magnification of 100 diameters in Plate III, Fig. 8, this group being on the extreme right of the latter. Opposite 12 is a group of four Betz cells arranged radially, the upper of the two in the middle being the largest, and the lowest next in size. Opposite 13 is a large solitary Betz cell, and above this and opposite 14 is a group of three Betz cells. Opposite 15 is another large solitary Betz cell, below which, and a little deeper in the cortex between it and the previously described group, is another Betz cell of medium size. Just above this is a group of two smaller Betz cells. Opposite 16 are three large cells in a group which are just to the left of Plate III, Fig. 8, and are intermediate in type between the well defined Betz cells and the ordinary large pyramidal cells, above this and opposite 17 is another group of four cells similar in type. Opposite 18 is a group of two Betz cells, one considerably larger than the other and nearer the surface of the cortex; in the same radial line is a medium sized solitary Betz cell. Just above this are five Betz cells opposite 19 and radially arranged in a row. Opposite 20 is a solitary Betz cell, and just above opposite 21 is a group of two Betz cells, one very large and the other quite small. Above this (to the left) are a number of pyramidal cells scarcely large enough to be considered as Betz cells, although as previously noted there are intermediate forms which might be considered as very small Betz cells or very large pyramidal cells. Opposite 22 are seen three Betz cells rather scattered, the one in the middle being at a deeper level of the cortex. Opposite 23 is seen a small solitary Betz cell, and opposite 24 is another solitary Betz cell which is on the anterior aspect of the gyrus. Between it and the last described cell are quite a number of large cells intermediate in type between the Betz cell and the largest pyramidal cells. Opposite 25 is still another solitary Betz cell. Then comes a region for a considerable distance in which are seen several large cells at this level, but hardly large enough to be considered as Betz cells until we reach that part of the cortex on the anterior aspect of the convolution opposite 26, where we see a group of two made up of a large Betz cell and a smaller one. Finally below this, at a considerable distance and opposite 27, is a group of three Betz cells, the one in the middle being large, and the other two small. It will be seen that the Betz cells are larger and more numerous on the posterior aspect of the gyrus than upon the anterior aspect. There are thirty-one groups of these cells in all, 13 of which contain but a solitary cell, so can hardly be considered as a group, though of course many of them form part of a group, the remaining cells being in another plane. Ten of these groups contain two cells each, 5 cells each, and but two groups have four cells each, and one group has five cells, making 61 Betz cells in all in this section. Beginning posteriorly and following around to the lowest group on the anterior surface, the number of cells in each group as seen in the same plane is as follows:—1, 2, 2, 1, 1, 1, 3, 2, 1, 2, 3, 2, 4, 4, 1, 3, 1, 1, 2, 2, 2, 1, 5, 1, 2, 3, 1, 1, 1, 2 and 3. These cells lie deeper in the cortex and thus at a greater distance from the surface in the posterior aspect of the gyrus than at the vertex or at the anterior aspect. Of course the number of cells in this one plane does not represent in every case all the cells in a group, but only those in this particular plane. As many of these groups are more or less spherical no one plane will show more than a part of such a group, and as stated above some of these solitary cells are the outer members of some of these groups.

Plate III, Fig. 8, as previously stated, shows the strip *a* of Plate III, Fig. 7, under a magnification of 100 diameters and in strong contrast to Plate II, Fig. 3, is to be seen the much larger number of neuroglia cells, the methylene violet stain seeming to have a stronger affinity for the neuroglia cells than the methylene blue stain. The cortex in this plate is also seen to be more condensed and not so deep as in Plate II, Fig. 3. The first layer measure but .20 mm. in thickness in contrast to .25 mm. in the above mentioned plate. In this layer neuroglia cells are more numerous, with a few scarcely scattered nerve cells only to be made out under high magnification, and similar to those found in the first layer in Plate II, Fig. 3. The second or pyramidal cell layer is of special importance and interest. It is deeper than the corresponding layer in Plate II, Fig. 3, measuring 1.75 mm. in thickness as contrasted to 1.40 mm. in the former. Here too in addition to the large and small pyramidal cells are found the giant pyramidal or Betz cells, irregularly distributed, singly or in groups, and at different levels, so that it is impossible to describe them as forming a distinct layer, although in this plate, with the exception of one cell, at *a* they appear to be placed in the lower part of this portion of the cortex now being described. In taking one section after another, however, it will be found that these cells are located at different levels, the majority being in the lower portion of this second layer. They furthermore have a tendency to be arranged in groups containing from two to ten cells, rarely more, and finally we find some sections containing no Betz cells at all. The pyramidal cells in the motor area are found to be larger in size on an average than those of the frontal region. The chromatic substance is not arranged in such large distinct bodies as in the Betz cells, but may be seen distributed in clumps of finer or coarser granules in different parts of the cell-body, especially about the nucleus and about the base, and at the beginning of the dendritic processes in the cell-body. Fig. M (M, of Plate III, Fig. 8), shows one of these cells under a high power, X1300 (No. 3, ocular, 1-12 inch oil immersion objective. Leitz). Here the finely granular chromatic substance is distributed in a ring about the nucleus, a clump is seen at the base to the left where a dendritic process is given off and also clumps are seen at the base of the dendritic process on the right and other clumps in the upper part of the cell-body and in the apical process. In addition to this, small sized linear and rounded chromophilic bodies are seen in the upper part of the cell-body and apical process and also in the dendritic processes given off at the base. The nucleus here is large, rounded, centrally placed, and contains a round, deeply stained, prominent nucleolus, surrounded by slightly stained homogeneous nuclear substance. In some cells the finely granular chromatic substance predominates in the cell-body, while in others the small chromophilic bodies are more prominent, whereas the majority contain both, as in the above described cell. In some of the smaller pyramidal cells the large nucleus almost fills up the cell-body, with but a narrow rim of finely granular chromatic substance surrounding the same and extending into the apical and basal processes. These latter cells would be classified under the group of karyochrome nerve cells; whereas the larger pyramidal cells with the stainable substance arranged in striæ in the same direction as the contour of the cell-body come under the head of the stichochrome nerve cells of the somatochrome class. Turning to the Betz cells and examining these under a high power (No. 3 ocular 1-12 inch oil immersion objective, Leitz. X1300) they are seen to contain numerous distinct chromophilic bodies varying in size and shape. In Fig. N (N, of Plate III, Fig. 8) the upper part of the cell is not included in this section. From below are seen two dendritic processes given off from the under side of the cell-body, that is having the cell-body from a lower plane than figured in the drawing, upon the right side; and one slightly larger process from the left side. These at intervals contain distinct chromophilic granules, for the most part linear in shape, with the long axis parallel to the axis of the process and extending for a considerable distance into the process—as far as they could be traced in the section. About the middle of the left dendritic process is seen a large irregular chromophilic body at a point where a branch is probably given off, as wedge-shaped chromatic masses are often found at the point of branching of a dendritic process, Nissl's so-called "wedges of division." Within the cell-body the granules are seen to be elongated, rounded or irregular in shape, the first mentioned predominating; some much larger than others, and with the general arrangement of the long axis parallel to the sides of the cell-body. The nucleus is large, well defined, and placed in the upper part of the cell-body and nearer the right than the left side. There is a well defined, deeply stained nucleolus centrally placed and surrounded by a homogeneous, slightly colored nuclear substance, with no well defined network. At the base of the cell-body, occupying a space somewhat larger than the cell nucleus is a deposit of yellowish pigment, elliptical in outline, and entirely displacing all other cell contents. No other pigment is to be found in this cell. The arrangement of the chromophilic granules in striæ in the same direction as the contour of the cell-body classifies it as a stichochrome nerve cell of the somatochrome class. Fig. o (o of Plate III, Fig. 8) shows another of these Betz cells examined under the same magnification of 1,300 diameters (No. 3 ocular, 1-12 in oil immersion lens, Leitz), in which the arrangement of the chromophilic bodies in striæ parallel to the contour of the cell-body is very well marked. This arrangement extends far out in the basal process and high up in the apical process. The nucleus is situated more on one side of the cell-body than the other, and the nucleolus is not included in this plane. There are three dendritic processes to be seen, the apical extending for a considerable distance towards the periphery, and gradually decreasing in size, and two basal processes, the one to the left unbranched, while the one on the right gives off a large branch shortly after leaving the cell-body, at which point *a* is one of the so-called "wedges of division" of Nissl. There is no pigment in this cell, but in the one seen just below this in Plate III, Fig. 8, marked P, a large mass of yellow pigment is seen above and to one side of the nucleus at the base of the apical process. There are but few chromophilic bodies in this cell compared to the two previously described, the chromatin being for the most part in the form of fine granules arranged in larger and smaller masses in different parts of the cell. The Betz cell seen lying just below and to the right of this latter, and marked Q in Plate III, Fig. 8, also contains a small mass of pigment poorly defined, and lying just below the nucleus at the base of the cell. The other Betz cells seen in this plate contain no pigment. While discussing the Betz cells, and before proceeding to the description of the third layer, several other photomicrographs will be described as illustrating the grouping and structure of these cells. The first of these, Plate IV, Fig. 9, is a photomicrograph magnified 14 diameters of a section in close proximity to that seen in Plate III, Fig. 7, of the same size as this latter and treated in the same way, excepting that methylene blue was used as the stain here. Opposite *a*, and enclosed in ink lines, is the segment of the section shown in Plate IV, Fig. 10, under a magnification of 100 diameters. The other layer is seen to vary in thickness, being much thicker at the posterior aspect, at *b*, than at the vertex or the anterior aspect.

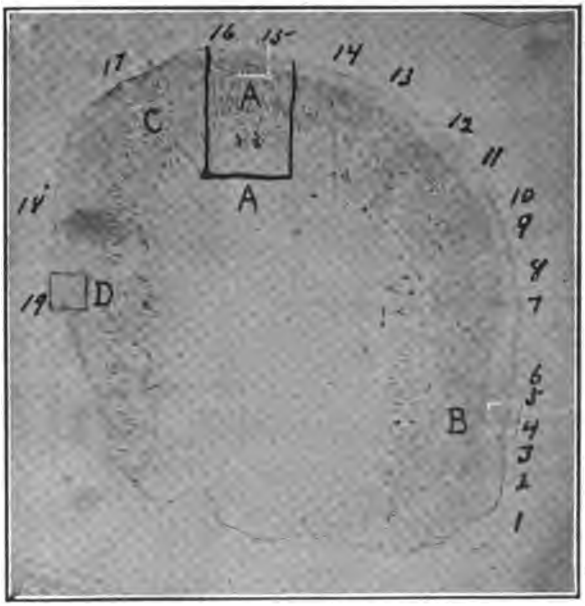


Fig. J.

At *c* and *d* the second layer is seen at the surface, due no doubt to injury in manipulation, probably more from pressure than actual tearing, as the surface is smooth and continuous at both ends, with the adjacent surfaces. No doubt artefacts similar to this, and of greater or less extent, must be carefully guarded against and considered in all work upon such a delicate and yielding tissue as the brain, especially when secured within a few minutes after death, as in this case, and before any post-mortem fixity results. The second layer shows the radial arrangement of the cells, but the most conspicuous feature is the distribution and arrangement of the Betz cells contained in the lower part of this layer singly or in groups of varying number and size. Below this the less conspicuous third layer is gradually lost to view as it merges into the white medullary substance. The arrangement of the Betz cells will be described in detail as photomicrographs of this magnification, 14 diameters, show their distribution especially well. Beginning at the posterior portion of the convolution below the letter *b*, the first large Betz cell is to be seen opposite the figure 1, and is solitary, whereas just above and opposite 2, is a group of four Betz cells all of about the same depth of the cortex. Above this, opposite 3, is a large solitary Betz cell. Opposite 4 is a group of three cells arranged radially above one another. Opposite 5 is another solitary Betz cell. Above it a small portion of a Betz cell is seen which lies mostly in another plane, while below it small portions of three Betz cells are seen lying for the most part in another plane. Above this, and opposite 6, is a group of two Betz cells at the same depth, and one just above the other. Between 6 and 7 are seen cells, two in number, which might be regarded as intermediate in character between the large pyramidal and Betz cells, having a similar arrangement of chromophilic substance, but of intermediate size. Opposite 7 is a group of two Betz cells, and opposite 8 is another group of two Betz cells somewhat nearer the surface, and just under this latter group and deeper in the cortex is a solitary Betz cell. Opposite 9 is a group of two Betz cells, only a very small portion of the upper cell being seen in this plane. Opposite 10, a portion of a solitary Betz cell is seen, and opposite 11 is still another solitary Betz cell. Opposite 12 is a group of three Betz cells, and just to the right of this and at the same level, is a part of a solitary Betz cell, while nearer to the surface and in the same radial line is still another small solitary Betz cell. Opposite 13 is a group of six Betz cells, two of which are large, and appear very prominently, the other four lying somewhat to the right, being smaller, but having the characteristic structure of the Betz cells. Opposite 14 is a solitary Betz cell, and next we come to the two groups seen under a magnification of 100 diameters in Plate IV, Fig. 10, and in this plate seen enclosed in ink lines and opposite figures 15 and 16. The first group, or that opposite 15, contains nine well defined Betz cells, while the adjacent group, opposite 16, contains six Betz cells, three large cells and the segments of three others, the greater part of these latter lying in another plane. Opposite 17 are four scattered cells at about the same depth of the cortex, and intermediate in size and structure, between the large pyramidal and the Betz cells. Opposite 18 is a large solitary Betz cell, above and below it being the cells intermediate in type just described. At 19 is a group of six, made up of one large Betz cell and five smaller ones about it. Above and below this are other pyramidal cells at this level, and of the above described intermediate type. There are in all fifty-nine of these Betz cells in this section, arranged singly or in groups; beginning posteriorly at 1, and proceeding around to the anterior and last group at 19, as follows: 1, 4, 1, 3, 1, 1, 3, 2, 2, 2, 1, 2, 1, 1, 3, 1, 1, 6, 1, 9, 6, 1 and 6, thus making twenty-three groups in all, eleven of which contain solitary cells, four contain two cells, three contain three cells, one contains four cells, three contain six cells, and one nine cells, in the plane of this section. Of these cells thirty contain distinct pigmentary deposits, these being practically all of the larger cells. Quite a number of the others in the plane of the main portion of the cell would undoubtedly also contain pigmentary deposits. The grouping, of course, is here described for only the plane of this section and the probability is that some at

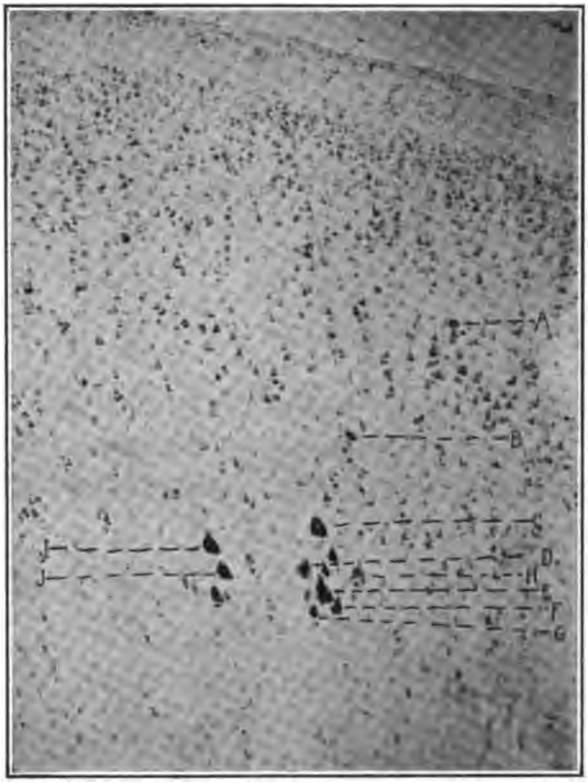


Plate IV, Fig. 10

least of these solitary cells are a part of a group,, the remaining cells lying in another plane. Furthermore, some of the groups described may contain additional cells lying in the plane above and below that of this section. Plate IV. Fig. 10, previously mentioned, has a magnification of 100 diameters, and includes the strip *a* outlined in ink in Plate IV. Fig. 9. Two groups of Betz cells are seen, one containing nine and the other six cells. Two other cells, *a* and *b*, though smaller yet in structure approach in character the Betz cells. It will be noticed that the cortex is not so deep in this plate, the first layer being but .175 mm. in thickness, and the second but 1.20 mm. in thickness in comparison to .20 and 1.75 mm., respectively, in Plate III. Fig. 8. It will be noted, however, that this strip was taken from the vertex of the section where the cortex is not so deep as on the posterior aspect from which Plate III. Fig. 8 was taken. These two groups of Betz cells are but 1.20 mm. from the surface of the cortex here in contrast to 1.70 mm. in Plate III. Fig. 8, showing the variability in thickness of the cortex in close proximity, as both plates were made from

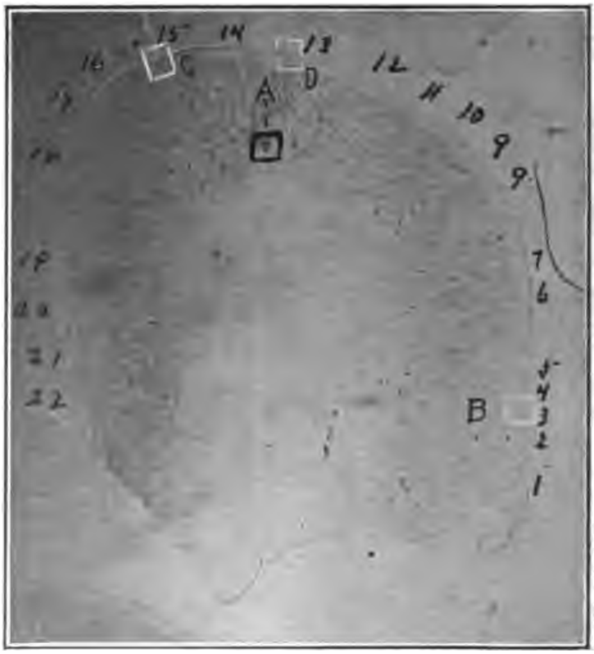


Plate IV, Fig. 11

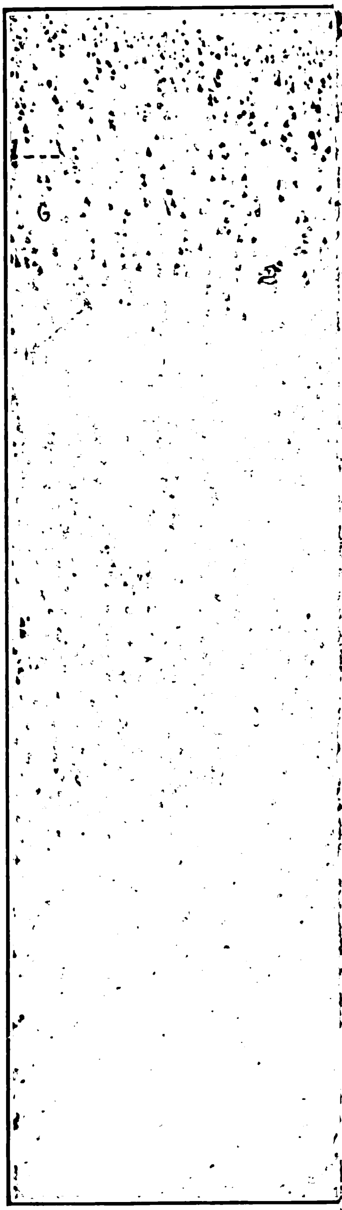
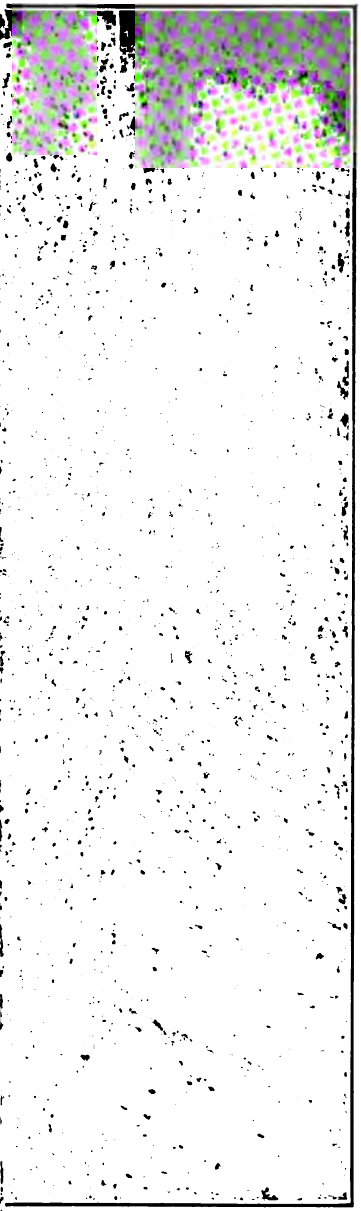


Plate II, Fig. 3.



ig. 21.

Plate VIII, Fig. 23.

sections cut from the same block which was not more than half a centimeter in thickness. The Betz cells here have the same structural characteristics as those described in Plate III. Fig. 8, and five of the cells, marked *c, d, e, f,* and *g,* of the larger group, and two of the cells, marked *i* and *j,* of the smaller group, contain distinct yellowish pigment. The larger of the two groups and containing nine Betz cells is made up of two very large cells cut in the plane of the nucleus, four smaller cells also cut in the plane of the nucleus, while the remaining three cells show only a small portion of the body of each cell here, the large portion of these latter being in another plane. Five of these nine cells contain deposits of pigment, and all show the chromophilic granules, being seen especially well in the cells marked *c, d, e,* and *h.* The cells in this group are quite close to one another and do not take up a very large space. The smaller group to the left contains six Betz cells, three large and conspicuous, and three others, one to the right and two to the left of these large cells which lie only partially in this plane and are small in size. Two of the three large cells marked *i* and *j* contain deposits of yellowish pigment at the base of the body of the cell, and all contain well defined chromophilic bodies. Plate IV. Fig. 11 is another photomicrograph of a section from the same block as that from which the sections represented in Plate III. Fig. 7, and Plate IV. Fig. 9, were taken. This section is 10 microns in thickness, stained with methylene blue and otherwise similarly treated as the above mentioned plates. This magnification of 14 diameters shows the outer layer to be fairly uniform in thickness, excepting at the points *b* and *c.* At *b* it is of relatively the same thickness as at the same point *b* in Plate IV. Fig. 9, about one and one-half times as thick as at *d* into which it gradually merges. At *c* where no outer layer is to be seen in Plate IV. Fig. 9, here the outer layer is distinctly seen but somewhat thinner than on either side, showing the artefact to be less in degree in this section. At *a* the group of cells represented in Plate V. Fig. 13, is seen, and each cell can easily be distinguished and counted under this magnification of 14 diameters. The second layer here is also especially interesting in respect to the size, number and distribution of the Betz cells singly or in groups in the lower portion of the layer near the transition into the third or spindle cell layer. This latter is less distinct and lost below in the medullary white substance in the interior of the gyrus. A detailed description of the arrangement of these Betz cells will be given in order to compare the same with those of Plate III. Fig. 7, and Plate IV. Fig. 9. Beginning with the first group at the lowest part of the section on the right, which is the posterior surface of the gyrus, opposite I, is seen a group of three large Betz cells. Above this some distance opposite 2, is a solitary Betz cell. Opposite 3, is another group of three Betz cells at a slightly greater depth. Above this and opposite 4 and 5, respectively, are large solitary Betz cells. Then there is a considerable interval where there are a number of large pyramidal cells but scarcely approaching the size of a Betz cell until the point opposite 6 is reached where a large solitary Betz cell is seen and just above it a group of two Betz cells somewhat smaller in size is to be noted. Opposite 7 is a solitary Betz cell at a greater depth. Just above this and slightly nearer the surface are several intermediate type cells arranged in two rows, one at a slightly greater depth than the other. Opposite 8 and at the same level as the preceding is a solitary Betz cell, and above this opposite 9 is a larger solitary Betz cell. At the same depth and slightly nearer the vertex is a group of two Betz cells, one large and the other small. About midway between these and the surface of the cortex are seen several very large pyramidal cells, the so-called intermediate type between the Betz cells or giant pyramidal cells and the ordinary sized large pyramidal cells. Opposite 10 is seen a group of two very large Betz cells just below and to one side of a large blood vessel. Just above this and nearer the surface is a small solitary Betz cell. Opposite 11 are three groups of Betz cells, the first containing three, the second two, and the third above and nearer the surface consisting of a large solitary Betz cell. Opposite 12 is a large solitary Betz cell, considerably nearer the surface than the groups just described. A group of two large Betz cells, one just above the other, is seen opposite 13. This brings us to the large group opposite *a* and seen under a magnification of 850 diameters in Plate V. Fig. 13, containing eight Betz cells which will be described in detail later when discussing this latter plate. To the left of this group opposite 14 are seen three Betz cells of small size and somewhat separated from one another. To the left of these at a short distance are seen two Betz cells, one above the other. Opposite 15 are seen two groups of two each, the cells in the first group being somewhat separated from one another and one above the other. Opposite 16 is a well marked group of four Betz cells, two being very large and the other two smaller in size. Below this and upon the anterior surface of the gyrus opposite 17 are two solitary Betz cells, one lower than the other and nearer the surface. Opposite 18 is still another smaller solitary Betz cell at a greater depth. For some distance below this no Betz cells are to be seen until opposite 19 is a solitary Betz cell. Just below this and slightly nearer the surface is a group of three of the intermediate sized cells. Opposite 20 are two large solitary Betz cells, the lower larger one being at a slightly greater depth. Then comes another region barren of these cells until the point opposite 21 is reached where a small solitary intermediate size cell is seen, and below this a group of two of these same cells is to be noted, one at a greater depth than the other. Opposite *22* is a very large solitary Betz cell. Just below this are two intermediate size cells, and still below this and at a slightly greater depth are three of these intermediate size cells. This includes all the Betz cells though, as above noted, here and there in different parts of this layer; but for the most part in the same relative position, are to be seen very large pyramidal cells approximating these Betz cells both in size and structure, and the writer thinks should be regarded as intermediate in type between the large pyramidal cells and the giant pyramidal or Betz cells,—a transition form, if they might be so called. There are twenty-eight groups of these Betz cells in all in this plate, varying in number from one to eight cells in each group, one to three being the usual number. Fourteen of these groups contain but one cell each, and these so-called solitary cells are usually of large size. There are eight groups containing two cells each, four groups of three cells each, but one group containing four cells, and one group containing eight cells. Taking the groups from the lower portion of the posterior surface in succession, as above described, and the number of cells in each of the twenty-nine groups is found to be as follows: 3, 1, 3, 1, 1, 1, 2, 1, 1, 1, 2, 2, 1, 3, 2, 1, 1, 2, 8, 3, 2, 2, 2, 4, 1, 1, 1, 1, 1, making a total of fifty-five Betz cells in the section, and thus averaging about two cells to each group. The number of groups is somewhat more than in Plate IV. Fig. 9, in which there were twenty-three groups containing in all fifty-nine Betz cells, and somewhat less than in Plate III. Fig. 7, where there are thirty-one groups containing sixty-one cells. It will thus be seen that the average number of groups and total number of cells in these three sections from near-by portions of the same block are fairly uniform in regard to the arrangement and distribution of the Betz cells. It is to be noted that more cells are found on the posterior aspect of the gyrus than on the anterior. In Plate III. Fig. 7, there are fifty cells arranged in twenty-five groups on the posterior aspect of the gyrus, with but four cells in two groups at the vertex, and seven cells in four groups on the anterior aspect. In Plate IV. Fig. 9 there are thirty-seven cells arranged in nineteen groups on the posterior aspect of the gyrus, fifteen cells in two groups at the vertex, and seven cells in two groups on the anterior aspect. Finally, in Plate IV. Fig. 11, there are twenty-seven cells arranged in seventeen groups in the posterior aspect of the gyrus, nineteen cells in six groups at the vertex, and nine cells arraned in six groups on the anterior aspect. It will thus be seen that most of the Betz cells are on the posterior aspect of the gyrus and gradually diminish in number upon approaching the vertex until upon the anterior aspect there are but few to be seen. Table III. shows the above results in tabulated form. Upon referring to this latter it will be seen that the average number of cells in each section is fairly uniform—55, 59 and 61 for the three sections; the grouping is somewhat more variable—23, 29 and 31, most of the cells singly or in groups of two or more being upon the posterior aspect and vertex. This corresponds to the findings of Lewis and Clarke ('78) who examined sections from various parts of the left anterior central convolution and the posterior portion of the two upper frontal convolutions on the same side in several human brains microscopically, and made diagrams of the distribution of the Betz cells at various points instead of employing photomicrographs showing the number, arrangement and relation to other elements of the cortex of these Betz cells as in this article. Plate V. Fig. 13 is a beautiful photomicrograph of a group of these Betz cells magnified 825 diameters and taken from the point marked *a* in Plate IV. Fig. 11. Here is seen a "nest," or group of these cells, eight

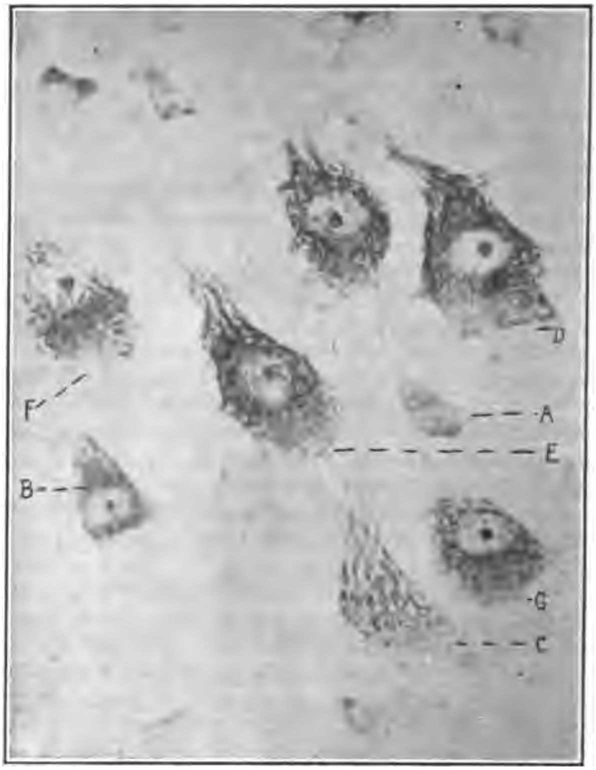


Plate V, Fig. 13

in number. All of these Betz cells contain pigment excepting the one marked *a* which in size is not much larger than one of the large pyramidal cells, but in structure resembles the Betz cells. This pigment, varying in amount, is situated at the base of the cell below the nucleus, excepting in the cell marked *b* where it is just above the nucleus. In the cell marked *c* it extends down into the large basal dendritic process, given off to the right for a short distance. In the cells marked *c, d, e, f* and *g*, the cell body appears of a lighter color in the photomicrograph where this yellowish pigmentary deposit is found. The cell marked *c*, sectioned outside of the plane of the nucleus, shows very well the general arrangement of the rounded or elongated chromophilic bodies in a direction parallel to the contour of the cell-body, thus classifying it with the others of similar type as stichochrome nerve cells of the somatochrome class. The cell marked *e* shows the general parallel arrangement of the chromophilic bodies in the apical process very well. The group, as a whole, is very compact and as each cell is cut in a somewhat different plane from the others most of the features of such a "nest" are thus brought out. Of course, there may be other cells in this group lying outside of the ones seen here —in the planes above and below this. Plate IV. Fig. 12 is a pho-

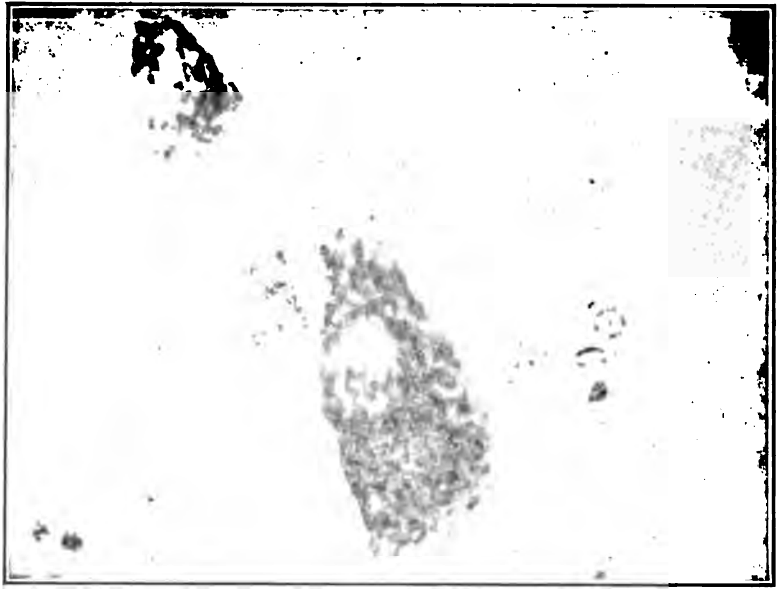


Plate IV, Fig. 12

tomicrograph of a Betz cell magnified 1,400 diameters from a section adjacent to and upon the same slide as that from which Plate III. Fig. 8 was taken. The section is, therefore, similar in shape to the one in Plate III. Fig. 7, and this cell is situated in a position opposite to the letter *d*, and in the lower portion of the second or pyramidal cell layer of this latter. The section is ten microns in thickness and stained with methylene violet. It can readily be appreciated from this photomicrograph that only an extremely thin plane can be seen under such a high magnification and not all of this even will be in sharp focus. The nucleus here is mostly below the plane of the section and the deeply stained nucleolus entirely so. Two dendritic processes are faintly seen coming off from the base of the cell, the one on the right but faintly shown as it is at a different level for the most part. The one on the left

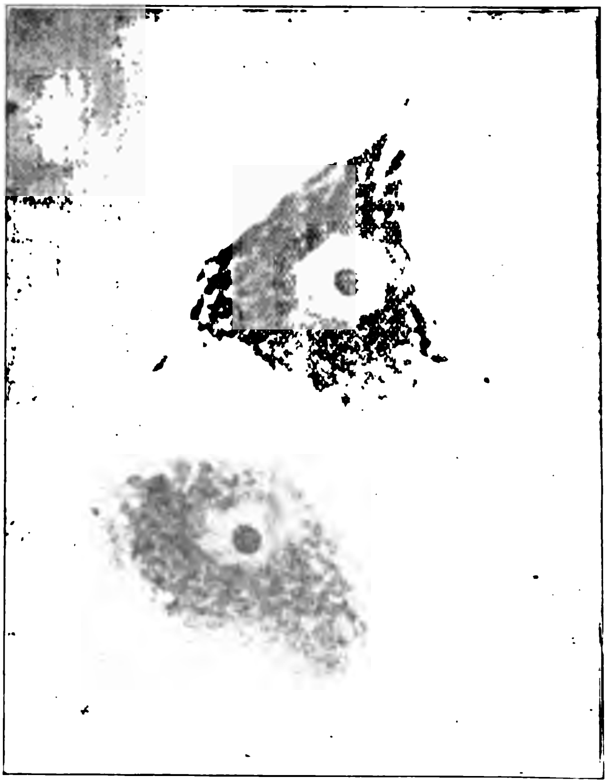


Plate V, Fig. 14

is somewhat more distinct and chromophilic bodies are found to enter the base and extend for a considerable distance into the same. These bodies of varying size and in general rounded or linear in shape, are closely packed in most parts of the cell-body, excepting at the base where a large deposit of yellowish pigment, taking up about the same area of space, as the nucleus, is to be found. These chromophilic bodies are arranged in general parallel to the contour of the cell-body and extend into both the basal dendritic processes and the apical process as far as they can be traced. A portion of another Betz cell smaller in size is seen above and to the left. About the larger cell are several neuroglia cells singly or in groups, and these are seen to be distinctly rounded or oval with usually a ring of chromatic substance of varying thickness at the periphery and several irregular deeply stained bodies in the center. Twenty neuroglia cells in all are seen in this plate. Plate V. Fig. 14, is a photomicrograph of the same magnification,—1,400 diameters,—as the preceding and taken from a section of the same block. The section of the same form and size as that seen in Plate III. Fig. 7, is ten microns in thickness, and stained with methylene blue instead of methylene violet, but otherwise treated in exactly the same way. One noticeable point about this plate is the absence of neuroglia cells, in marked contrast to Plate IV. Fig. 12, indicating again that the neuroglia cells do not retain the methylene blue stain with the same intensity as when similarly treated with methylene violet. Here is seen a group of two Betz cells, the upper one, at this plane broadly pyramidal in shape, and the lower one presenting an irregular spindle shape. This latter, however, is only the base of the cell, the process above and to the left and that below and to the right being basal dendritic processes, and the apical dendritic process extending upward beyond the base of the upper cell in a different plane. There are two other Betz cells just above and slightly to the right of these cells in the section, not shown in the plate, thus making the group consist of four cells in all. Both of these cells are cut in the plane of the nucleus and large, rounded, deeply stained nucleolus. The nuclear body also contains some chromatic substance arranged in an indefinite network. At the base of the lower cell and extending into the large basal dendrite to the right as far as it is seen in this section is a distinct mass of yellowish pigment displacing the other cell contents. In the plate it is of a lighter color than the rest of the cell. Above this in the cell-body about the nucleus, and extending into the other basal dendrite, are numerous rounded and linear chromophilic bodies. The upper cell shows three dendritic processes going off from the base of the cell-body on the right side, and two from the left side. These processes and the large apical process contain mostly linear-shaped chromophilic bodies, arranged in distinct striæ, parallel to the contour of the same. This arrangement also holds good at the periphery of the cell-body, the striæ there too being parallel to the contour of the same, but about the nucleus the chromophilic bodies are so closely packed that it is more difficult to make out this parallel arrangement. There is no pigmentary deposit in this cell. No neuroglia cells are to be seen in this plate, the methylene blue stain being used as stated above. Plate V. Fig. 15, from the same region and under the same magnification, 1,400 diameters, from a section ten microns in thickness and stained also with methylene blue, is an exceptionally fine photomicrograph of one of these

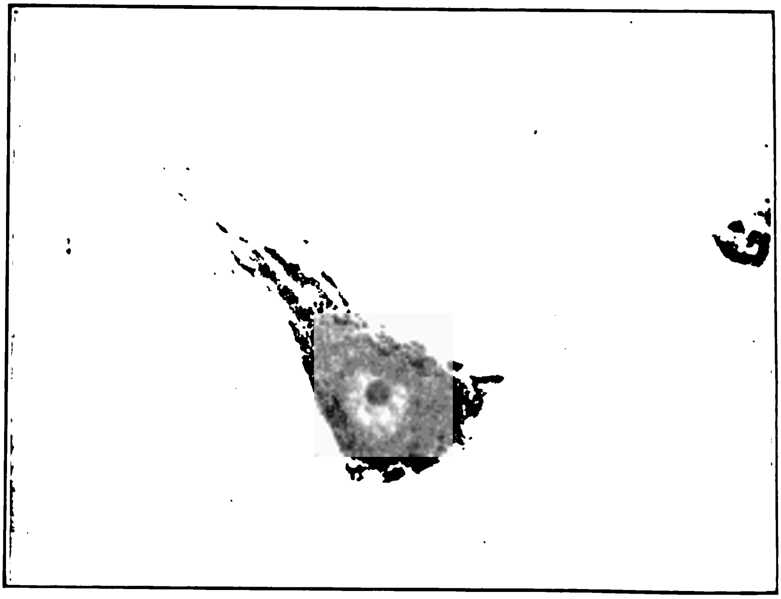


Plate V, Fig. 15

Betz cells, inasmuch as it shows the processes, especially the apical process, as far as the edge of the plate. It is usually difficult to obtain such an extent of surface of a cell in the same plane under such high magnification even when the microtome cuts in exactly the plane of the long axis of the cell. These dendritic processes are seen to be of no mean size, especially the apical process and the chromophilic granules extend far up into the same. Three processes are seen given off from the base, one at each angle, and the third descending from the center. The nucleus is distinct, centrally located, and contains a large nucleolus surrounded by an irregular chromatic network. The chromophilic granules are larger and smaller rounded and irregularly spindle-shaped granules in the triangular-shaped body of the cell, approaching to a more linear form in the processes. This is a stichochrome nerve cell of the somatochrome group.

Turning again to Plate III. Fig. 8, the third layer will now be described. This is but .70 mm. in thickness as compared to 1.20 mm. for the same layer in Plate II. Fig. 3. The nerve cells in this layer are similar in form and structure to those found in the above mentioned plate. The neuroglia cells are much more numerous in all the layers than in Plate II. Fig. 3, due in large part, the writer believes, to the difference in the staining method. Capillary vessels, showing the walls made up of a single layer of nucleated flattened cells, are seen in several places, especially in the lower layer. Counts were made of the nerve and neuroglia cells in different regions in the manner previously described, using the same method and magnification. Counts of the nerve cells in eight different fields from various portions of the second and third layer were made, each field containing thirty-six squares, so that 288 squares were examined. There were found to be an average of .95 nerve cells to each square millimeter of the ocular net-micrometer, which multiplied by 100 gives 95 as the average number of nerve cells found to the square millimeter in the second and third layer of the cortex in this section. The first layer contained so few scattered nerve cells that no field was included for this portion of the cortex, as some fields here would contain no nerve cells whatsoever and would thus greatly reduce the general average where the Nissl stain is employed. On this account the nerve cell counts in this and all subsequent sections, as well as that of Plate II. Fig. 3, include only the second and third layers of the cortex. The nerve cells are thus fewer in number than in Plate II. Fig. 3, but they average larger in size, especially in the region of the large pyramidal and Betz cells; thus the frontal convolutions seem to to contain more nerve cells than the central convolutions.

Neuroglia cell counts were made from three fields of thirty-six squares each or 108 squares in all from all three layers of the cortex. The average number of neuroglia cells was found to be 2.93 for each square millimeter of surface of the ocular net-micrometer, which multiplied by 100 gives 293 as the average number of neuroglia cells in each square millimeter of surface of the cortex from this section. The number in each square here varied from none in some squares to fourteen in one of the squares. The next highest number was eight, several contained seven, but most of them contained from one to four. Counts were also made from a section of the same thickness, ten microns, and from the same block as the preceding, but stained with methylene blue instead of methylene violet. With this exception the technique was exactly the same in the two preparations. Counts were made from six different fields of thirty-six squares each, and the average number of nerve cells to each square mm. of the ocular net-micrometer from various parts of the second and third layers was .94. This result multiplied by 100 gives 94 as the average number of nerve cells to each sq. mm. of surface of the section as compared to 95 in Plate III. Fig. 8, a remarkably small difference of only one cell to each square millimeter, both counts being made, the one entirely independent of the other. Counts were made from seven different fields of 36 sq. mm. each from various parts of all three layers of the cortex to determine the average number of neuroglia cells. The average number to each square millimeter of the ocular net-micrometer was found to be .84, which multiplied by 100 gives 84 as the average number of neuroglia cells to each square millimeter of the section as compared to 293 in Plate III. Fig. 8, where the methylene violet stain was the stain used. This further confirms the statement previously made that methylene violet has a greater affinity for these neuroglia cells and the latter retain this dye much more tenaciously than they do the methylene blue in the subsequent differentiation, whereas the nerve cells retain either dye about the same. The same fact is well

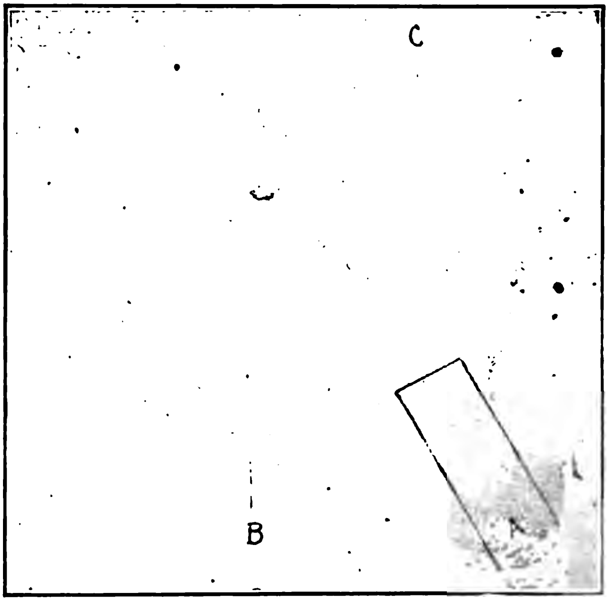


Plate VI, Fig. 16

brought out in Plate IV. Fig. 12, and Plate V. Fig. 14, previously described. In the latter two, Betz cells are seen under a magnification of 1,400 diameters. The stain used here is methylene blue, and no neuroglia cells are to be seen. In Plate IV. Fig. 12, of the same magnification, but stained with methylene violet, twelve of these neuroglia cells are seen in the immediate vicinity of this large Betz cell. Sections were also prepared from blocks taken from the middle and lower thirds of this same convolution,—the anterior central,—and it was found that the general type of cortical lamination and histological structure remained the same. Plate VI. Fig. 16, is a photomicrograph of a section taken from a block including the entire anterior central convolution for a distance of .4 mm. in its lower third and is representative of the entire lower portion of this convolution. The shape of the convolution is seen to have undergone considerable modification as compared to the upper third, seen in Plate III. Fig. 7. This section is ten microns in thickness, fixed in alcohol 95 per cent., stained with methylene violet, the other technique being similar to previously described sections. The segment of cortex shown in Plate VI. Fig. 17, under a magnification of 100 diameters, was taken from the point indicated by the ink lines here and opposite *a*. It is seen from the mechanical arrangement of the cortex to be at the most acute angle and where the same is thickest. At the opposite less acute angle the cortex is not quite so thick, and intermediate between these angles, and also on the anterior and posterior surfaces the cortex is still thinner. It will be noted that the depth of the sulcus anteriorly at *c* where it passes over into the third frontal convolution and is thus adjacent to the motor speech area, is more shallow than the posterior sulcus or fissure of Rolando. The first layer is irregular in depth, somewhat thicker at the angle opposite *a*, and still thicker at *b* and *c*, whereas at the vertex it is thinner. The general radial direction of the cells from the medullary central white substance to the surface is fairly well seen here. In the second layer the most noticeable feature is the entire absence of the Betz cells which are so marked in the upper portion of the convolution. Some very large pyramidal cells, intermediate in size to the Betz cells and the ordinary larger pyramidal cells, are to be seen in this layer however, as will be noted in the following (Plate VI. Fig. 17): This layer merges into the third or irregular cell layer, which latter is gradually lost in the white central substance of the gyrus. At the vertex in the cortical layer the section is cracked; this being an artefact resulting from manipulation. The outer layer is seen to be also torn in places from the same cause. Plate VI. Fig. 17, is taken from the strip indicated in ink in Plate VI. Fig. 16, and is magnified 100 diameters. The first layer is .25 microns in thickness and similar in structure to the same layer in the upper portion of the convolution already described. In the second layer, however, the greatest difference is found. The large Betz cells practically disappear in the lower third of this convolution, although large pyramidal cells simulating them in general structure, but lacking the size, are found in the middle portion of this layer. In this lower third of the convolution the second layer as seen in this section is 1.50 mm. in thickness, and the large pyramidal cells are seen arranged in irregular groups in its middle portion. Below them is seen a region *a* averaging .25 mm. in thickness in which small pyramidal cells are almost exclusively found. Below this larger pyramidal cells are seen, but not in great numbers. The divison between this and the lower or spindle cell layer is not well marked, the latter measuring .90 mm. in thickness. The cells here are irregularly polygonal or spindle shaped and do not differ from those in the same region in the upper part of this convolution. The total thickness of the cortex at this point is 2.65 mm., exactly the same as of the section shown in Plate III. Fig. 8. Counts of the nerve and neuroglia cells were made in this region in exactly the same manner as in the previously described regions. This section, as above stated, is ten microns in thickness and stained with methylene violet and in seven fields of 36 sq. mm. each from various portions of the second and third layers, the average number of nerve cells to each square millimeter of surface of the ocular net-micrometer was found to be 1.08, multiplied by 100 gives 108 as the average number of nerve cells to each square millimeter of surface of the section in the second and third layer. The nerve cells in the first layer were so few and scattered that no counts of the same were made. In eight fields of 36 sq. mm. each from various portions of the three layers of the cortex in this section the average number of neuroglia cells to the square millimeter of surface of the ocular net-micrometer was 2.48, multiplied by 100 gives 248, as the average number of neuroglia cells to each square millimeter of surface of the cortex of this section. Here again it will be seen that the methylene violet stain shows a larger number of neuroglia cells—248, while the nerve cells—108, are but slightly in excess of the average number in the two previously described sections of the upper portion of the anterior central convolution, and slightly less in number than in the sections described from the frontal convolution (Table II.).

Passing now to the region posterior to this, we come to the posterior central convolution of this central or motor region. This posterior central convolution is regarded as a transition region between that anterior to the fissure of Rolando, or motor region, and that posterior to the same region, or sensory region of those authors who divide the cortex into a sensory and motor type of cortical lamination. The first and third layers here are practically the same as in the region anterior to the fissure of Rolando. The second layer, however, lacks the regularity of the corresponding layer in the above mentioned region, but as will be seen in the plate (Plate VII. Fig. 19), there is nothing new or different in this layer from the corresponding layer in the plates already described. The large Betz cells seen only in limited portions of the region just anterior to this and contained in but a very small portion of this so-called motor region are replaced here by some smaller sized, irregularly situated pyramidal cells, singly or in groups, and of similar internal structure, and mostly to be found in the middle and lower portion of the layer. As typical of this posterior central convolution, a section was taken from a block including the middle portion of this convolution in its entirety for a distance of 4 cm. (5, Plate I. Fig. 1). The section is 6 2-3 microns in thickness, fixed in 95 per cent. alcohol, and stained with methylene blue, technique otherwise similar to all other sections. Plate VI. Fig. 18, is a photomicrograph of this section, magnified 14 diameters. The convolution here presents a peculiar mechanical formation, causing a great variation in thickness of the cortex in different portions, being over twice as thick at *b* as at the point *a*, from which latter point Plate VII. Fig. 19 was taken. This condi-

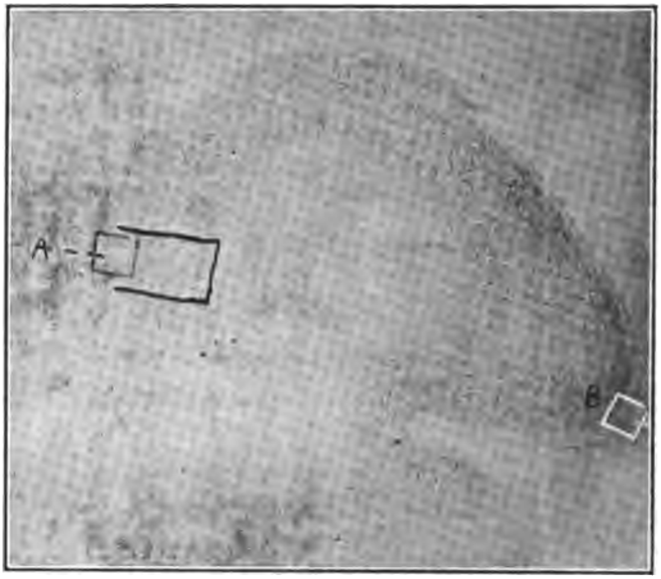


Plate VI, Fig. 18

tion is not an artefact, as under high magnification no indication of disturbance of the relations of the cells is to be observed. At the vertex, however, there is an artificial break in the first and upper part of the second layer at two points. The first layer is seen to vary in thickness, being thicker at the acute angle below *b*, and gradually becoming thinner as it approaches the vertex, to become again thicker below *a*. The general radial direction of the cells is well seen, especially at the vertex and anterior aspect, as at *a*. At *b* it is not quite so well made out, but exists to a fairly well marked degree when examined under a higher power. Pyramidal cells of considerable size are seen in some parts of this layer, more especially in the middle portion, but in no case do they approach in size to the Betz cells, nor are they for the most part situated in the same portion of the cortex. The third layer is thin and soon lost to view in the medullary white substance. Plate VII. Fig. 19, is a photomicrograph of the strip *a* enclosed in ink lines in Plate VI. Fig. 18, and magnified 100 diameters. The first or upper layer at this point is somewhat irregular in contour and averages .20 mm. in thickness. It contains neuroglia cells and some scattered nerve cells similar in appearance and structure to those described in Plate II. Fi. 3, for the same region. The second or pyramidal cell layer is especially interesting, owing to the irregularity of the distribution of its cells. At the upper portion the small pyramidal cells are seen closely aggregated together. Below this these cells become more scattered, and irregularly interspersed among them are medium sized and larger pyramidal cells. Some of the larger of these have a tendency to be arranged singly or in groups and have an internal structure similar to the giant pyramidal or Betz cells found in parts of the anterior central convolution and in the posterior part of the first frontal convolution. The chromophilic granules are, however, of smaller size though similarly arranged in some of the cells. About the middle of this layer, opposite *a*, is seen another region in which the small pyramidal cells predominate, and in which there are scarcely any larger pyramidal cells. Just below this again are seen large pyramidal cells arranged singly and in groups, forming the lower portion of this second layer which measures about 1.45 mm. in thickness and below gradually merging into the third or spindle cell layer. In addition to the spindle cells there are many irregular polygonal cells founds in this layer which at this point is about .60 mm. in thickness and only partially shown in the plate; the upper two-thirds being seen here. The lower third, not shown, is similar and gradually merges into the white medullary substance. The entire cortex is thus 2.25 mm. in thickness at this point, whereas, as above mentioned at point *b* in Plate VI. Fig. 18, it is over 5 mm. in thickness. Taking into consideration the variations found in different parts of the same area, often quite near together, and considering the general type of arrangements, it does not seem to the writer that this area varies to a very marked degree from the general type, or at least to such an extent as to be especially marked off from the other regions of the cortex. There is certainly a greater variation between different regions of the anterior central convolution as in the upper and lower portions, where some parts have no Betz cells and others have them variously distributed, as well as other differences, than between this region and the frontal region as a whole. Those who separate the cortex into two types,—an anterior or motor type, and a posterior or sensory type,—regard this convolution as the transition region between the two. An examination of sections, both anterior and posterior to this convolution, shows that the same general plan of arrangement is carried out in all parts of the external surface of the cortex with only minor differences which do not seem sufficient to make such a marked distinction applicable anatomically, and certainly not histologically. The size and form of the cells and their general arrangement differs but slightly from that of the region anterior and posterior, with the exception of the Betz cell groupings in localized portions of the former.

Nerve cell and neuroglia cell counts were made with the ocular net-micrometer here in this region in the same manner as in the other previously described sections. Eight fields of thirty-six square millimeters each, from various portions of the second and third layers of the cortex, were examined and 185.5 nerve cells were found to be the average number for each square millimeter of surface of the section. In the same manner the neuroglia cells were counted in eight fields from all three layers of the cortex and 109.2 neuroglia cells were found to be the average number for each square millimeter of surface of the section in the cortex. Here methylene blue was the stain employed with the resulting low average number of neuroglia cells, whereas the average number of nerve cells is found to be higher than in any of the previously described sections anterior to this region, and it will be seen subsequently that it is also higher than that found for the region posterior as well although the section is 6 2-3 microns in thickness, and some already described are 10 microns in thickness (Table II.). This, however, bears out the statement previously made that sections between six and ten microns in thickness contain practically all nerve cells that can be seen in one plane.

*Parietal Region.*—Posterior to the region just described we come to the parietal region, made up of the superior parietal, supra-marginal, and supra-angular convolutions. The general arrangement of the layering of the cortex is fairly uniform throughout this region and the section represented in Plate VII, Fig. 20, from a block taken from the supra-marginal convolution is typical of this region (7, Plate I, Fig. 1). The block was fixed in Van Gehuchten's fluid, stained with methylene violet, and other technique as for previous regions. The actual size of this section is 7 mm. in its greatest width, and 8 mm. in length, and it is 6 2-3 microns in thickness. As seen in the photomicrograph, magnified 14 diameters, the convolution is wider above at the surface than deeper down in the sulcus. At the point *a* is seen the position of the strip from which the photomicrograph represented in Plate VII, Fig. 21 was taken. At *b* and *c*, owing to the greater angle, the cortex is considerably thicker than at *a*. The shape of the convolution here is seen to be different again from any of the preceding, thus giving a somewhat different mechanical arrangement, with a broad flattened vertex and shorter anterior and posterior aspects. At the lower portion, on the right, a part of the cortex was cut away in removing the block. In the vicinity of *a* there are some minute artefacts, causing an irregularity of the surface here, and small fragments are partially torn away at two points. The first, or superficial layer, is fairly uniform in thickness, excepting opposite *a*, which will be discussed more fully in describing the next plate (Plate VII, Fig. 21), and upon

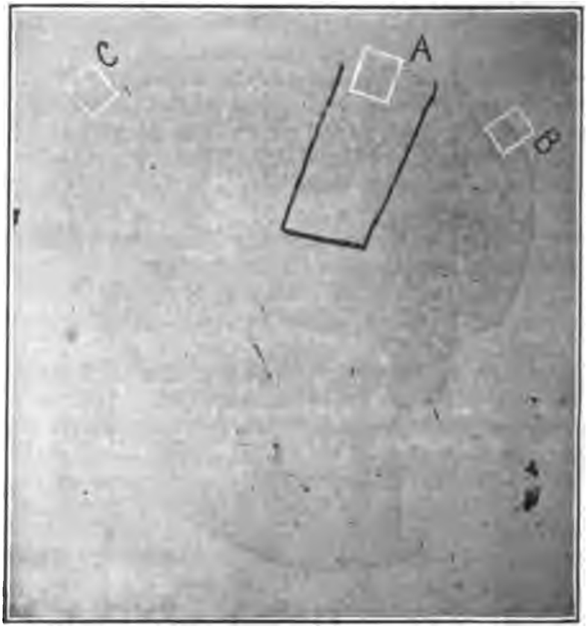


Plate VII, Fig. 20

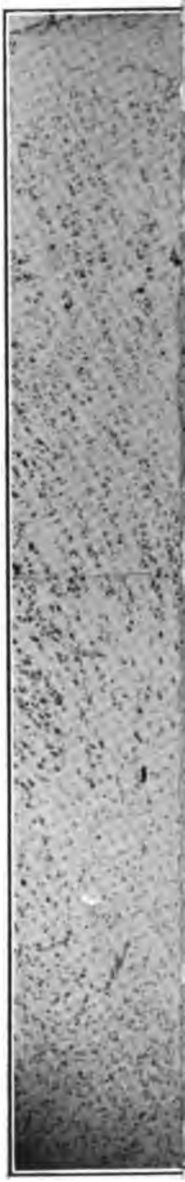


Plate IX, Fig

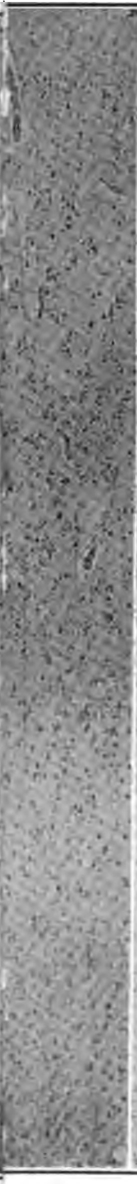


Fig. 45.

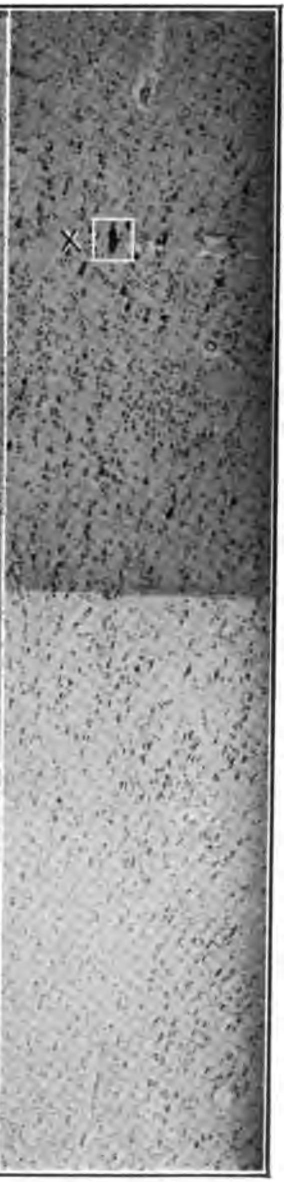


Plate XV, Fig. 47.

the posterior and anterior aspects, where this layer is slightly thicker. The radial arrangement of the cells of the second layer is well shown, and the cortex is seen to be thicker at the angles *b* and *c*, as before mentioned. Throughout the middle and lower part of this layer well developed pyramidal cells are seen, some almost approaching in type small Betz cells. The third layer presents nothing unusual, and gradually passes over into the white medullary substance. Plate VII, Fig. 21, shows the strip indicated in ink opposite *a* in Plate VII, Fig. 20, under a magnification of 100 diameters. The first or superficial layer is seen to be irregular at this point, about 20 mm. in thickness at the right of the plate, then increasing rather suddenly to .25 mm. for a short distance, when the outline suddenly descends, decreasing the layer at this point to .15 mm. It then gradually increases until, at the left of the plate, it is .22 mm. in thickness. The extreme thickness near the center, although the surface is only slightly torn at several points and scarcely noticeable, yet has the appearance of an artefact, due no doubt to the unequal pressure in the manipulation of removal, when the brain was in its natural soft condition, prior to any post-mortem changes. A short distance below the surface at this point the cortical substance itself is seen slightly broken, thus confirming the artificial nature of the irregularity and increase in depth. The nerve and neuroglia cells are similar to those found in this layer in the regions anterior to this. The second or pyramidal cell layer is 1.60 mm. in thickness, and contains small pyramidal cells exclusively in the upper portion. Very soon, however, larger pyramidal cells appear, and at about the middle of the layer are the greatest in size and most numerous of the layer. Some of these large pyramidal cells in point of size and structure appear intermediate between the large and giant pyramidal or Betz cells. They also tend to be arranged singly or in groups, with smaller cells about them. Just below the middle of this layer, at *a*, is found a region .20 mm. in thickness, conspicuous by the almost entire absence of large pyramidal cells, and made up for the most part of small pyramidal cells. Below this large pyramidal cells again appear, intermingled with the small pyramidal cells, and the layer gradually passes over into the lower or spindle-cell layer. The pyramidal cells have the same internal structure as those in the region anterior to this, in the largest ones the chromophilic granules simply being larger and more conspicuous; thus these cells come under the same classification, according to Nissl, as those in the frontal and central convolutions. The third or spindle-cell layer is .70 mm. in thickness, and not distinctly separated from the layer above. The arrangement of the cells and their form and size, as well as the internal structure, is in no way different from the corresponding layer in the cortex anterior to this. The total thickness of the cortex here is then 2.50 mm., being .25 of a mm. thicker than in the section of the posterior central convolution previously described, .15 mm. less in thickness than the section shown in Plate III, Fig. 8, from the anterior central convolution, and .35 mm. less than in the section of the first frontal convolution shown in Plate II, Fig. 3. The variation in thickness is thus seen to be slight and no greater, and even not so great as can be found in contiguous portions of any one convolution. In other parts of the parietal region the arrangement of the cortical lamination is similar to that described above with minor modifications, due to differences in shape, etc., of the convolutions. Here nerve cell and neuroglia cell counts were made in the same manner as in previous regions, and the average number of nerve cells to the square millimeter of surface of the section was found to be 104, whereas the average number of neuroglia cells for the same area of surface of the section was 180.90. This is the lowest average of nerve cells in any region of this brain, with the exception of the upper portion of the anterior central convolution. The methylene violet stain makes the average number of neuroglia cells quite high, however, though considerably less than in the upper portion of the anterior central convolution.

*Temporal Region.*—Below the parietal region just discussed we come to the three temporal convolutions—the first, second and third. Here the general arrangement of the cortical laminations and the cell structure is practically the same in the three convolutions, and bears a striking resemblance to that found in the frontal region. A block was taken from the anterior portion of the first temporal convolution near the apex (6, Plate I, Fig. 1), the entire width of the convolution and for a distance of .4 cm. The actual size of the section from this block represented in the photomicrograph (Plate VII, Fig. 22) is 8.5 mm. at the widest point, and 8.5 mm. in length on the right, and 6 mm. in length on the left, and it is 6 2-3 microns in thickness. The block was fixed in Van Gehuchten's fluid, stained with methylene blue, all the other details of technique being similar to that employed for previously described sections. This photomicrograph is magnified 14 diameters, and here the shape of the gyrus is seen to be somewhat similar to that in Plate. VII, Fig 20, but broader above, and also with a broader base. The vertex is flattened similarly, resulting in the more acute angle at *b* and the slightly obtuse angle at *c*, at which points the cortex is thicker than at the vertex or the anterior or posterior aspects. The cortex is seen to be uniformly thicker along the surface, opposite *d*, than upon the opposite aspect at and above and below *a*. The first or superficial layer is thicker at *c* than in other portions, and is somewhat broken in places along the vertex. The second layer varying in thickness, being thickest at the points mentioned above, where the entire cortex is thickest, shows the radial arrangement of the cells very well, especially at the vertex and opposite *a* and *b*. The larger nerve cells appear quite conspicuous in the middle and lower portion of this layer in places. The third layer is quite uniform, disappearing in the white medullary central substance. Plate VIII, Fig. 23, shows the strip *a* of Plate VIII, Fig. 22 at a magnification of 100 diameters. Here the differentiation has been carried to about the same stage as in Plate II, Fig. 3, and the paucity of neuroglia cells is equally as apparent. Again the general arrangement of the cells in the two plates is remarkably similar. The first or superficial layer is here .25 mm. in thickness, and contains, for the most part, neuroglia cells and a few scattered nerve cells. The second or pyramidal cell layer measures 1.65 mm. in thickness, as compared to 1.40 mm. in Plate II, Fig 3, being thus slightly thicker than in the latter. The cells are arranged practically the same, however, the small pyramidal cells above intermingled with larger pyramidal cells in increasing numbers deeper down in the layer. Here again, at a point somewhat deeper than in Plate II, Fig. 3, is to be found a region at *a*, of the same thickness—.30 mm., where small pyramidal cells are found almost ex-

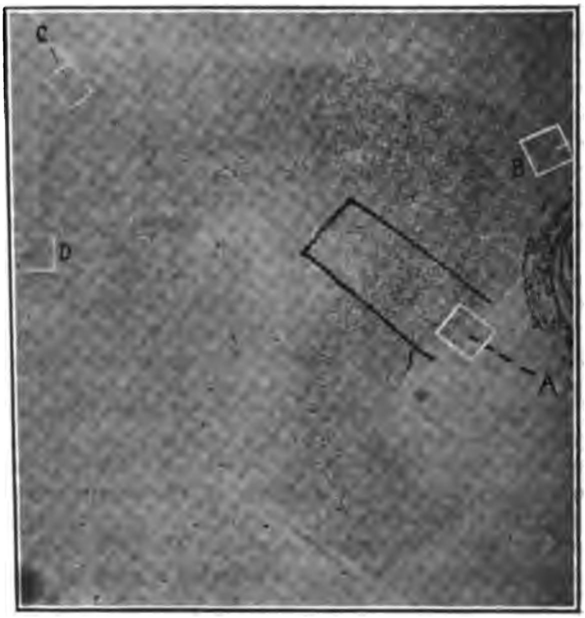


Plate VII, Fig. 22

clusively, followed again by a region containing large pyramidal cells in considerable numbers in addition to small pyramidal cells. This layer finally passes over into a narrow (.40 mm. thick) third or spindle-cell layer. This layer is also similar to that of Plate II, Fig. 3, with the exception of its depth, the latter having a spindle or irregular cell layer, measuring 1.20 mm. in thickness. As will be seen by referring to Plate I, Fig. 2, the strip *a* represented in Plate II, Fig. 3, was taken from the point of greatest curvature, where the cortex is usually the thickest, whereas Plate VIII, Fig. 23, was taken from the point *a* shown in Plate VIII, Fig. 22, from the side of the section where the cortex is thinner. The entire depth of the cortex is found to be 2.30 mm., as compared to 2.85 mm. in Plate II, Fig. 3, a difference of .55 mm. There are no larger pyramidal cells here intermediate in size between the Betz cells and the ordinary large pyramidal cells, as were noted singly or in small groups scattered in the central and parietal convolutions already described, and, as we shall see, also occur in the occipital convolution. The internal structure of these cells and their classification are similar to that for the cells described in Plate II, Fig. 3. Nerve and neuroglia cell counts were made from this section, which, as above mentioned, is 6 2-3 microns in thickness and stained with methylene blue. Seven fields of 36 sq. mm. each gave an average of 146 nerve cells to each square millimeter of surface of the section, and eight fields of the same size gave an average of 131.90 neuroglia cells to each square millimeter of surface of the section. It will be seen upon comparing these figures with those from sections of regions more anterior and above, that they show a larger number of nerve cells than in the sections from the regions anterior to the fissure of Rolando, but a less number than that for the posterior central convolution, and more than from the parietal region. The neuroglia cells are about the same in number as in Plate II, Fig. 3 of the frontal region.

(*To be continued.*)

# Periscope.

## ARCHIV FUER KRIMINAL ANTHROPOLOGIE

(Vol. 12, 1903, Nos. 2, 3.)

1. Daktyloscopie. C. Windt.
2. Rendering Latent Impressions Visible. F. Paul.
3. Impressions of Elmira. Wintry.
4. Medical Experts in Psychiatry. J. Berze.
5. Infanticide by Cold Bath. R. Mothes.
6. Alcoholism and Testimony. R. Pollitz.
7. Signs of Death by Hanging. Strassmann.
8. Falsification of Seals. W. Schütze.
9. Preliminary Cross-Examination. H. Gross.
10. Anatomical Seat of Criminal Tendencies. P. Näcke.
11. Legal Views of Crime. R. Mothes.
12. Reform Movement in Criminal Procedure. G. Lelewer.
13. Legal Science in Greenland. D. B. Oefele.
14. Reading of Thoughts. H. Schneickert.
15. Superstition: Fortune-Telling and Quackery. W. Schütze.

1. *Daktyloscopie.* C. Windst points out the great value of the papillary lines at the finger tips for identifying criminals. The pattern remains the same throughout life, and even if a portion of the skin is torn off accidentally, the new-formed skin will not alter the design. Even when bodies have been lying in water for weeks, they can be readily identified by impressions from the fingers. All patterns may be divided into four classes, which have received the following names: Bow (arcus), loop (lasso), whirl and combined pattern. A very extensive system of cataloguing the impressions from all ten fingers by means of arbitrary letters and fractions is described in detail. Compared with anthropometry, the method seems to be equally reliable and much simpler; it can be applied just as easily to children, females and cadavers. Occasionally the criminal himself helps in his identification by leaving behind an impression of his fingers on glass, etc. By suitable methods these impressions can be rendered more visible and permanent.

2. *How to Render Visible Latent Impressions.* From F. Paul's article it appears that many chemicals can be used to render visible and permanent accidental impressions of fingers on glass, paper, etc. The simplest method is to expose the object to the fumes of iodine. Silver-nitrate in 8 per cent solution also gives good results if the paper is then exposed to sunlight and treated with hyposulphite of soda. Other available substances are ink, many powders in moderate subdivision (calomel), permanganate of potash. With glass, the fumes of hydrofluoric acid may also be employed.

3. *The Reformatory in Elmira.*—Wintry has visited Elmira and speaks very favorably of the management of the institution and the régime among the inmates. He believes that from 75 to 80 per cent of these are completely cured.

4. *Difference of Opinion Among Experts in Psychiatry.*—J. Berze states that it is oftimes impossible to estimate correctly the mental capacity of a patient. This refers especially to those cases which are not really mentally diseased but merely exhibit a deficient mental development. The individual equation plays a prominent part here, since we possess no normal

standard by which to gauge the degree of intelligence. Even in acquired conditions there may be a psychical weakness rather than definite and characteristic symptoms, so that the difference from the normal is quantitative rather than qualitative. Almost insurmountable difficulties may be encountered in distinguishing between temporary and permanent aberrations in neurasthenia, hysteria and epilepsy. Last, but not least, the expert will often be in a quandary to express an opinion in psychopathic deficiency, that intermediary condition which has underlying it neither a normal nor an abnormal mind. There is no proper place for these patients as yet; they belong neither in an insane asylum nor in prison, yet the expert is often forced to choose between the two. It is difficult to find a remedy for the difference of opinion so frequently encountered; even if separate institutions existed for these patients there would still be occasion for dispute, but the consequences would not be so serious.

5. *Attempt at Infanticide by Means of a Cold Bath.*—R. Mothes briefly records the case of a woman who had purposely left her infant in a cold bath. The court acquitted the woman of attempted murder, but recommended her punishment for the affliction of bodily injury which might have resulted fatally.

6. *Testifying Upon Alcoholic Disturbances Before Court.*—Pollitz relates how difficult it may sometimes be to properly class alcoholic mental disturbances with reference to the ultimate psychical derangement which accompanies them. There is a tendency on the part of the laity to look upon alcoholism more as a moral defect than a disease, and, in consequence, the judge and jury are frequently inclined to condemn too harshly. The matter is frequently complicated by the fact that it is difficult to make the proper diagnosis, especially if the expert sees the patient after weeks or months of enforced temperance. Alcoholic psychosis may be divided into four classes, viz.: Delirium tremens, acute hallucinatory insanity, the acute pathological intoxications, and, lastly, chronic delirium. Of these, delirium tremens forms the prototype; it is that peculiar condition where the afflicted is fully aware of his own condition, but mistakes completely his surroundings. Prominent symptoms are optical delusions, marked suggestibility and motor unrest and a good recollection of the details of previous attacks. In the second type the delusions are more markedly of an acoustic character, the patients are better oriented, and the entire course is more chronic. Pathological intoxications are accompanied by a clouded consciousness. The recollection of the details is almost entirely lost, and sleep always follows. Illustrative cases of these three conditions are given.

7. *Signs of Death by Hanging.*—Strassmann had occasion to do an autopsy on an old man who first attempted to hang himself and then fired a bullet into his brain. As in a previous case recorded, tears in the mucous membrane at the laryngeal orifice were found.

8. *Methods Employed in the Falsification of Seals.*—A detailed article by W. Schütze, stating how signatures, seals, etc., are commonly altered by criminals.

9. *Value of Preliminary Cross-Examination.*—This article of H. Gross is directed against the suggestion to do away with the customary preliminary cross-examination. When properly conducted by an experienced judge it is the most valuable of the three commonly employed in Germany.

10. *The Anatomical Seat of Criminal Tendencies.*—P. Näcke does not believe with Lombroso that criminals and epileptics possess a prominent median occipital groove secondary to hypertrophy of the worm of the cerebellum. Prominent anatomists have shown that this condition is found almost as often in the brains of normal individuals (in 2 to 3 per cent), and hence cannot be looked upon as an atavistic change. Despite the many writings of Lombroso, the real anatomical seat of criminal tendency is as unknown to us as it ever was.

11. *Incorrect Legal Views as a Cause of Crime.*—R. Mothes mentions several peculiar notions on legal points prevalent especially among the lower classes. They often constitute reminiscences of former laws, and not infrequently lead to or justify crime.

12. *The Reform Movement in the Trial of Criminals.*—G. Lelewer discusses the contemplated reform in criminal practice, and advises against too great leniency.

13. *Legal Science in Greenland.*—The inhabitants of Greenland still possess very primitive ideas concerning law. According to D. F. B. Oefele, murder is very rare since the national character is peaceable and abhors dispute. A common practice, however, is to hasten the death of the old and dying. Married life is rather loose, and it is considered more of an advantage than a disgrace to possess children born out of wedlock. Disputes are usually settled by means of the so-called "drum dance," where the one who makes most noise is generally the victor.

14. *Reading of Thoughts.*—H. Schneickert has been present at several seances and has found that the thoughts to be transmitted are always communicated to the medium by a carefully studied and often complex system of signals. These are passed at moments when the attention of the public is diverted. Criminals frequently have a similar mute language, and partners should always be kept at a distance from each other, and all motions of their hands, face, etc., should be carefully watched.

15. *Superstition, Fortune-Telling and Quackery.*—The superstition of the lower classes has frequently ruined reputations and destroyed family ties. In the case cited by W. Schütze a perfectly innocent man was accused by almost all his acquaintances of arson because a "wise man" had thrown suspicion upon him. It is often very difficult to punish these quacks. In this case it took one and a half years to gather sufficient evidence.

O. HENSEL (New York).

### ARCHIVES DE NEUROLOGIE

(Vol. 15, 1903, No. 89, May.)

1. A Study of Mania. SOUKHANOFF—GANNOUCHKINE.
2. New Observations on the Neurosis of Anguish. P. HARTENBERG.
3. Physiopsychology among Religious Orders. BINET—SANGLÉ.
4. Critical Review of Stigmata of Degeneration.

1. *A Study of Mania.*—Drs. Soukhanoff and Gannouchkine of University of Moscow founded their study upon material accumulated in the psychiatric clinic of Moscow. The authors have been able to classify the "observations" of all patients, both interne and externe during fifteen years from 1887 to 1902. The total number of patients were 4,434, of whom 2,840 were men and 1,594 were women. Among these, they recognize mania in but 40 cases, of whom 16 were men and 24 women. In the category of mania, the authors exclude from consideration all cases where, before or after the onset of mania, a depressive state was noted; these they classify as circular psychosis. All cases are also excluded where mania appears as a complication of general paralysis, a senile dementia, etc. "The above data indicate very clearly that mania is a very rare psychic malady."

Women are prone to suffer from mania more often than men, and if the number of màle and female alieneés were equal, the ratio of men to women affected with mania would be as 1 to 2.68. Melancholia, particularly in women, appears as one of the most frequent of mental maladies; occurs almost seven times as often as mania. These views, they say, are those of other authors. Figures show that mania manifests itself most often between the ages of 16 and 25 years. The age of earliest access, 13 years; latest at 66 years.

2. *New Observations upon Neuroses of Anguish.*—Hartenberg, after

Freund of Vienna, and upon his own studies, classifies the subject: (1) General irritability, including often auditive hyperesthesia; (2) A state of habitual anxious expectation of some evil which threatens; (3) Crises of acute agony, beating of the heart, etc.; (4) Equivalents of crises, represented by nocturnal fright, with distressing dreams, etc., variations of general nutrition, with periodic fattening or emaciation; (5) Phobies and obsessions.

The first observation cited is that of a married woman, Madame L., twenty-six years of age, mother of a child of four and one-half years. She was in great mental distress, the origin of which was as follows: While absent from her home, "a friend of the house, made courtship to her and engaged her in a flirtation sufficiently advanced. There was great reciprocal attraction; the situation became perilous for the honor of the young woman, when suddenly the gallant friend, seized with remorse, put an end to their relations. They almost ceased from seeing each other." This, instead of calming her for the future, induced bitter remorse. She was in continued inquietude. A thousand questions arise in her mind. "Does she still love her husband? Her child? She is a great criminal. She ought to be punished. What misfortunes will happen to her, how escape them?" She weeps, is in despair, and calls for death to deliver her from her martyrdom. Somatic troubles accompany the mental state, etc. The author institutes a treatment tonic and reconstituent. While she improves in bodily health, sleeps, etc., the obsession, fear of punishment with scruples, reproaches, ideas of suicide, continue, and after six months the mental troubles have subsided but not disappeared. The neurasthenia is rapidly cured; the neurosis of agony is not amended till much later.

The author next propounds, with qualified approval, an hypothesis of Freund that neuroses of anxiety are caused by insufficiency of satisfaction of sexual want. He reports in his own practice the case of a widow, who afterward remarried, which gave some evidence of Freund's opinion. He also reports in much detail a case of the neurosis of anxiety produced by the emotions of automobilism. In 1899, M. F. commences automobiling, at first with a tricycle, then with a voiturette, and lastly with a carriage of six horse power. In May, 1899, he journeys from Nice to Macon, with four halts. The first three days pass without incident in spite of the great fatigue produced by the close attention required in guiding the carriage. The fourth day, in getting out at Neufchateau for luncheon, he finds himself greatly indisposed. He experiences severe pain in the epigastric region, like that in extreme emotion. His teeth chatter, he shivers, has violent beating of heart, and is assailed with fear of sudden death. In October following, after three days' journey in the automobile, he dines with good appetite and sleeps at Avignon. Towards eleven o'clock he is awakened with an atrocious terror. It seems to him that it is the end, that his heart will break; that he will instantly die—all this subsides in half an hour. A medical consultation reveals no heart lesion, and attributes the symptoms to a gastric state. M. F. has given up the automobile. *A propos* of this: on page 480 of this number, there is a report of a meeting of the Society of Hypnology and Psychology, at which there were remarks upon the psychology of speed.

Hachet-Souplet. Whoever is in a situation to increase at will his speed is entirely carried away by the intense impressions which he experiences; he loses self-mastery; he is intoxicated; there are born in him feelings which he has not in a normal state; pride, combativeness, anger, hatred, spitefulness, violence. These feelings vary in form and degree, according as it concerns the driver of an omnibus, the coachman on his cab, the horseback rider, the automobiliste.

Berillon. There is a great analogy between the euphoria of speed and that of morphine. Those who allow themselves to be carried away with speed-making, for its own sake, without any end of utility, are most often degenerates, deprived of power of doing things in moderation, have lost empire over themselves and contend in a fury against the slightest obstacles.

Paul Magnin. When we come to the question of speed, of tobacco or of alcohol, we have an affair of individuals, who in use,' are unable to keep from abuse; are in effect deprived of all power of moderation, to the point of losing the instinct of self-preservation. One physician thought that speeding a horse involved a difference, as it was control of an intelligent being, to which replies,

Lux. In Algeria I have been a witness of Arab fantasias. They are demi-fools, intoxicated by the dust, transported by a savage fury; have no longer any sense of moderation. Their euphoria resembles that of automobilists, and the horses even are likewise seized with it.

The author states, in summing up, that we may consider the clinical syndrome of the neurosis of agony as real and generally accepted at this day. But it remains not less true that there are some cases, where the affection as described exists in a pure state, without any participation of neurasthenia or of melancholia, and which spontaneously or under the effect of care is cured entirely, without ever turning into asthenic neurosis or into psychosis of anxiety.

3. *Physio-psychology Among Religious Orders: Nuns of Port Royal.*—The author continues and concludes his studies of the seventeenth century monks and nuns of Port Royal, France. After giving minute biographies of several, he sums up by saying that he finds a neuropathic and religious heredity in one of the nuns and, in two, a religious heredity. Of three others, one was suffering from dysentery and suppurating hepatitis, and obtained relief in her troubles by auto-suggestion; the second was in general bad health; at twenty-seven had vomitings; at thirty-two, spitting of blood; the third also was in weak health, and died after five days of "oppression in the breech," with continuous fever. Of the four, of which we know the age at death, the first died at fifty-one, the second at nearly seventy-four, the third at sixty-eight, and the fourth at eighty; the average of the four was sixty-eight.

As to character, one passed as intelligent, another said she was "poor in spirit" (Query: Does the author know of the beatitudes?), two presented a predisposition to sadness, one to weakness and fear; all were of hypersuggestibility; three had a certain suggestive power.

4. *Stigmata of Degeneration.*—This number contains a review of certain articles by René and Henri Larger on the "Obstetric Stigmata of Degeneracy." Besides physical and mental stigmata there are also obstetric stigmata of degeneracy as seen in anomalies of gestation, such as sterility, twin-bearing, miscarriage, ectopic pregnancy, all the anomalies of the placenta, of the membranes, of the cord, all abnormal presentations, etc. These views are contested by Poiak and the accoucheurs: First observation—First husband normal; presentations normal in "OIGA"; second husband degenerate, presentation of the face. Second observation—First paramour normal, two successive presentations normal in "OIGA"; second paramour degenerate, presentation by the breech; the second paramour is a married man who has, simultaneously by his wife, a child born also by the breech. Third observation—Husband degenerate, three pregnancies with, each time, presentation abnormal with puerperal convulsions. The wife takes a paramour perfectly normal; three new accouchments, all normal in "OIGA," and without puerperal eclampsia. Richards, Amityville.

### MONATSSCHRFT FUR PSYCHRIATRIE UND NEUROLOGIE

(Vol. 13, 1903, No. 5, May.)

1. The Aphasic Symptom-Complex. E. STORCH.
2. Contribution to the Knowledge of Transcortical Aphasia. M. BERG.
3. Contribution to the Further Knowledge of the Richness of the Cerebral Cortex of Human Beings in Myelinated Nerve Fibers. E. L. F. S. BRÜCKNER.
4. The Pathology of Paralysis of the Peroneus. S. DAUS.

1. *Aphasia.*—Storch commences his article with a careful analysis of psychical perception. This is of psychological rather than of medical interest. He mentions, however, as one of the most important facts, that attention to any part of the body diminishes the way of irritability of the motor cortical cells that cover that part. Speech differs from the visual perceptions inasmuch that it is wholly dependent upon hearing. He analyzes the acoustic perceptions. He draws a sharp distinction between musical perceptions and phonetic perceptions, the two being entirely independent. (The paper is still unfinished.)

2. *Transcortical Aphasia.*—The essential symptoms of transcortical aphasia are paraphasia, paragraphia, and echolalia. The variation in the anatomical changes found in the cases that have hitherto been reported indicate that it is impossible to localize the lesion that gives rise to this condition. Berg tabulates eighteen cases that he has obtained from the literature, including one of his own. (The paper is still unfinished.)

3. *Myelinated Fibers in Cortex.*—Brückner continues his article upon the medullary fibers in the cortex of an eighteen-year-old Suaheli. He gives elaborate tables showing the results of his painstaking measurements, and a wealth of anatomical details that unfortunately cannot be reproduced in an abstract. He compares the results with those obtained in other human brains, particularly with that of a German child. Curiously enough the former in some respects was equally as well developed. In other respects also the brain resembled the infantile German type, particularly in the superior development of the right hemisphere.

4. *Pathology of Peroneus.*—Daus calls attention to the frequency with which the peroneal nerve is paralyzed. He classifies these as follows: (1) Congenital; (2) acquired during extrauterine existence. These may be: A. Mechanical Causes.—I. Involving the central organs; (1) external injuries either to the head or back; (2) internal mechanical injuries: brain hemorrhage, brain tumors, or lesions or tumors of the cord. II. Affecting the peripheral and peroneal tract; (1) external cutaneous injuries. (a) stab, blow, or gunshot wounds; (2) burning; (2) injuries as a result of fractures or luxations; (3) rupture of the nerve, (a) simple over-stretching, (b) over-stretching as a result of tumors; (4) pressure, (a) pressure during sleep, (b) pressure during narcosis, (c) ligation, either external, as from a bandage, or internal as the result of scars or callus, (d) compression, (e) in deformities of the knee, (f) paralysis during birth either of the mother or child, (g) tumors; (5) miscellaneous, (a) paralysis as a result of overexertion, (b) the hysterical paralysis of Charcot, (c) paralysis due to fright, (d) the paradoxical contraction of Westphal, (e) paralysis due to habit of Erb.

B. As a result of toxic causes.—I. Directly toxic; (1) chemical: alcohol, lead, arsenic, mercury; II. parasitic, (a) acute infectious disease: diphtheria, pertussis, exanthemata erysipelas, typhoid fever, pneumonia, influenza, gonorrhea, syphilis, beriberi, and malaria; (b) chronic infectious diseases: tuberculosis and leprosy; III. indirectly toxic: Chemical: Diabetes, gout, rheumatism, skin diseases, and ascending neuritis.

C. As a symptom or complication of organic disease of the central nervous system.—I. In tabes, in tabo-paresis, in dementia paralytica, multiple sclerosis, antero-poliomyelitis, and the peroneal type of progressive muscular atrophy.

D. In old age.

E. Of unknown cause.

Daus quotes articles from the enormous literature on this subject, illustrating each of these forms. (The paper is still unfinished.)

(Vol. 13, 1903, No. 6, June.)

1. The Capacity of the Skull (The Cranial Cavity) in Nursing Infants and Older Children. H. Pfister.
2. Disturbances in Sensory Perception in Cases of Mental Disease. W. v. Bechterew.
3. The Aphasic Symptom Complex. E. Storch.
4. Contribution to the Knowledge of Transcortical Aphasia. M. Berg.

1. *Skull Capacity.*—Pfister has determined the capacity of the cranium by means of water, according to his own method, in 77 male infants, and 77 female infants, ranging in age from thirteen days to eight years. The variability in the size of the cranium in both sexes is considerable. In the second year there may be a difference of as much as 200 cm., although the average is only 948 cm. in males and 907 cm. in females. The average result for a certain period is as follows:

I. To the end of the first month: Male, 415 cm.; female, 414 cm.
II. To the end of the third month: Male, 503 cm.; female, 574 cm.
III. To the end of the sixth month: Male, 651 cm.; female, 577 cm.
IV. To the end of the tenth month: Male, 769 cm.; female, 684 cm.
V. To the end of the fifteenth month: Male, 899 cm.; female, 883 cm.
VI. To the end of the twenty-first month: Male, 1,030 cm.; female, 895.

As a result of his studies he reaches the following conclusions: First, the cubic capacity of an infantile skull less about 6.5 per cent, gives approximately the capacity of the cranium in the living individual. Second, the capacity of the cranium is larger at all ages in males than in females. The difference is less at birth and increases with age. Third, before the ninth month of life is completed about one-third of the total increase in the capacity of the cranium over that at birth, has occurred. Fourth, at the end of 2.5 years about two-thirds of the total increase in the capacity of the cranium has occurred. It is not known when the head ceases to enlarge. The individual variations in size are very considerable.

2. *Sensory Perception in Mental Disease.*—Von Bechterew reports some curious cases in which patients had strange disturbances of the psychical sense. A girl of twenty years, although touch, pain and temperature senses were not distinctly abnormal, had a hallucination that her head, arms, legs and even the body were missing. The condition appears to be one of purely psychical anesthesia. In another instance, an army officer had such a hyperesthesia for the word "blood" that when it was spoken, he was sometimes thrown into convulsive states. In still another case a patient had hallucinations of hearing associated with certain sensory disturbances.

3. *Aphasia.*—Storch concludes his article upon the aphasic symptom complex. He discusses perception of letters, of words, and particularly the relations of these perceptions to the various forms of aphasia. He uses the term glosso-psyche to indicate the entire apparatus used in the formulation of letters and the spelling of words. He devotes considerable time to the discussion of spelling, and finally concludes with a classification of the various forms of aphasia.

*A.*—Those without essential participation of the stereopsychic region; —intelligence and tact; (1) The proglossopsychical aphasias. The lesion lies peripherally to the glossopsychical fields (the subcortical forms, (a) subcortical sensory aphasia, or pure word-deafness: the lesion being in the left temperosphenoidal lobe; the glossopsychic region is slightly or not at all injured; the invalid can read and write; (b) subcortical motor aphasia: aphemia and pure word-deafness: the lesion being in Broca's region. The glossopsychic region is almost intact; the patient understands speech, can read and write, but cannot speak. (II) Glossopsychic aphasias, (a) cortical sensory aphasia, the lesion having separated the glossopsychic region from the peripheral organs of hearing, and also injured the glossones. In addition to word-deafness there may be paraphasic manifestations, and disturbances of reading and writing; (b) cortical motor aphasia: the lesion not only separates the glossopsychic region from the phonetic nuclei in the medulla, but also has injured the latter. The understanding of speech and the ability to read and write are involved according to the extent of the lesion; (c) pure glossopsychic aphasia: in which only the elements of glossopsychic region are involved: therefore the understanding of speech, spontaneous speech, repetition and reading and writing are injured; (d) total aphasia in which the whole glossopsychic region is destroyed.

B.—Forms of aphasia with involvment of the stereopsychic region; (1) Pure stereopsychic forms, that is, certain varieties of transcortical motor and sensory aphasia, pure word-blindness, various form of agraphia and psychoses; (2) mixed stereoglossopsychic aphasias.

4. *Transcortical Aphasia.*—Berg continues his article with a long description of his own case. A man for forty-nine years had shown symptoms of mental impairment for several months. The essential features of the case were that voluntary speech was preserved as far as the formation of words was concerned, but the language was completely paraphasic and incohert. The understanding for words and simple sentences was preserved, but more complicated sentences produced hopeless confusion. Voluntary writing showed pronounced paragraphia; the understanding of writing for words and simple sentences was preserved. The patient could repeat, could read, could copy, and could even write from dictation. The repetition of simple words was done intelligently. If more complicated sentences were given the paraphasic phenomenon sometimes appeared. In reading, copying and in writing from dictation the patient understood nothing. The echolala was typical. The patient was able to identify objects by sight and touch. Intelligence, memory and observation were all diminished. The case, therefore, represents a form of transcortical aphasia, partly motor and partly sensory in character, and the lesion is probably a diffuse lesion in the brain. JOSEPH SAILER (Philadelphia).

### ARCHIV FUER PSYCHIATRIE UND NERVENKRANKHEITEN

(37 Band, 1903, I Heft.)

1. Concerning Korsakow's Symptom-Complex. E. MEYER and J. RAECKE.
2. The Pathogenesis of Amyotrophic Lateral Sclerosis. H. HAENEL.
3. A Contribution to the Pathology of Huntingdon's Chorea. E. STIER.
4. A Contribution to the Pathology of Chorea Minor. C. HUDOVERING.
5. The Simple Demented Form of Dementia Praecox. O. DIEM.
6. The Value of the X-Rays in the Treatment of Diseases of the Spinal Cord. E. VON LEYDEN and E. GRUNMACH.
7. Further Contributions to the Pathology of Sensory Aphasia. A. PICK.
8. The Occupation Neuroses of Telegraphers. E. CRONBACH.
9. Notes on the Criticism of Dr. M. Fekch on Two Cases of Cerebellar Palsy in Children.

1. *Korsakow's Symptom-Complex.*—An investigation of the question whether multiple neuritis may be followed by definite mental symptoms. The history of eight cases is given in detail. One improved greatly so that he was practically cured; one was improved; two remained unimproved and four died. Of the four cases that went to autopsy one showed fresh degen-

eration in the crossed pyramidal tracts, another had minute hemorrhages into the gray cerebral substance, a third case showed an old apoplectic scar in the right thalamus and in the pons and the fourth had a large sarcoma in the right frontal lobe. The authors conclude that their cases show positively that Korsakow's symptom-complex is not a disease *sui generis,* also decidedly not an alcoholic psychosis, but is present also in those diseases in which irreparable or severe destruction occurs in the central nervous system.

2. *Amyotrophic Lateral Sclerosis.*—The patient was a woman forty-five years old, with good heredity, previously in good health, married, with four children. Alcoholism and syphilis were excluded. Two years previously it was noticed that the speech of the patient was awkward, also there began at that time a slowly increasing weakness in the right side, first in the arm, then in the leg. Speech was later suddenly lost although at the time of examination it was slow and indistinct, but intelligent, without paraphasia. The clinical diagnosis was bulbar paralysis and amyotrophic lateral sclerosis. Shortly before death atrophy of the small muscles of the hand, particularly on the right, was distinct. Babinski was marked on both sides. Patient became uncleanly in her habits. At necropsy the kidneys were found small, granular and with shrunken capsules; the heart was hypertrophied and dilated. A microscopical examination was made of the cerebrum, spinal cord and muscles of the tongue with Nissl, Weigert, Van Gieson and carmine stains. No new facts are added. The author inclines to the belief of a vascular toxemia in his case. A satisfactory bibliography and an excellent photograph are appended.

3. *Huntingdon's Chorea.*—One case is reported in which Nissl's thionin stain was used. Comparing his findings with those of other authors he makes the deduction that Huntingdon's chorea is always dependent upon an inherited asymmetry of the cerebral cortex. The asymmetry can frequently be seen by the unaided eye. The disease begins in middle life with proliferation of the neuroglia in the motor centers. This proliferation may be either diffuse or circumscribed and is most marked in the second and third cortical layers; that is, the layers of the small and medium-sized pyramidal cells. At the same time the blood vessels become diseased, showing outwandering of the lymphoid cells into the perivascular spaces and occasionally hemorrhage. Almost always the small and medium-sized ganglion cells become degenerated throughout, while the large ganglion cells in the innermost layer remain intact.

4. *Chorea Minor.*—A case in which bacteriological examination of the blood and a part of the cord gave negative results. Interesting findings were inflammatory changes in the central nervous system, particularly marked in the motor pathways in the pons and medulla; rounded bodies, presumably colloid, which lay in the neghborhood of the blood vessels and also free in the brain substance; light grade of ependymitis and leptomeningitis and a disruption of the cell nuclei in the cortex particularly in the pyramidal cells of Ammon's horn. (Four beautiful plates show these conditions clearly.)

5. *Dementia Praecox.*—Nineteen carefully recorded cases are added to the literature. The author makes a strong plea for the retention of the name to express a disease entity which when carefully studied shows a well-defined clinical picture.

6. *X-Rays in Cord Disorders.*—An inspiring article on the value of the Roentgen Rays in the diagnosis and treatment of affections of the spinal column with illustrative cases. Thirty patients in all were examined; in twelve the diagnosis was simply confirmatory, but in the other eighteen the diagnosis was first positively made or at least further amplified by the rays. In ten of the eighteen cases primary disease of the bony column was followed by paraplegia. These ten cases comprised compression of the dorsal medulla by tuberculous bony deposits; compression of the dorsal medulla

by kyphoscoliosis, two cases; two new growths, one carcinoma, the other sarcoma; two cases of cervical myelitis from fracture, and one case of bulbar paralysis, probably due to dislocation of the third and fourth cervical vertebræ. In the eight following cases the previous diagnosis was verified and much relief afforded by suitable treatment. One was dorsal myelitis, one, syphilitic pachymeningitis and myelitis, and one, lumbar myelomeningitis, and two cases of tabes dorsalis. In all of these distinct osteoporosis was diagnosed by the rays. In another case diagnosed as tabes an osteoarthropathy of the spinal column was further diagnosed, and in still another case of tabes with arthropathies of both knees the diagnosis of osteoarthropathy of both knees was made and an osteoporosis of the spinal column was shown.

7. *Sensory Aphasia.*—Will be abstracted when completed.

8. *Telegrapher's Neuroses.*—But sixteen previously reported cases could be found. The author first reviews the literature and then gives the history of his own case, which showed nothing of particular note. In eleven of the total cases it is interesting to observe that the affection began in the spring months. A neuropathic tendency is shown by nervous weakness in the immediate family in six of the cases, by apoplexy in three cases, by gout in two cases and by stomach trouble with convulsions in one case. Complications with other diseases was noted in nine cases. The treatment promises little. The therapeutic measures advised are great, change of scene, baths, cold water cure, faradism, galvanism, massage, gymnastics, change of occupation or of the method of work. Of these measures massage gives the best results, while change of occupation did not give the benefit one would expect. The use of the Morse instrument intermittently with the Hughs instrument is advised as a prophylactic measure.

9. *Cerebellar Palsy.*—This is a caustic reply of Dr. Sommer to the criticism of Dr. Felch. (36 Bd. 3 Hft, S 895 et al.)

F. Witmer (New York).

### AMERICAN JOURNAL OF INSANITY

(Vol. 59, 1903, No. 4, April.)

1. A Contribution to the Study of the Blood in Manic-Depressive Insanity. J. S. Fisher.
2. The Hallucinatory Delirium of Acute Alcoholism. C. S. Walker.
3. The Progress of Psychiatry in 1902. A. R. Urquhart.
4. Puerperal Insanity. R. Jones.
5. The Care of the Insane. C. G. Wagner.
6. Tent Life for the Insane. O. J. Wilsey.
7. A Graded and Systematized Plan of Outdoor Exercise for the Demented Insane. C. L. Carlisle.
8. The Final Chapter in the History of an Extensive Injury to the Head. H. P. Frost.
9. Therapeutic Notes. R. Dewey.

1. *Blood in Manic-Depressive Insanity.*—Owing to no account being taken of data that may have an influence on the condition of the blood, the author of this paper made his observations at the same time of the date under uniform relations as to food, baths, bodily temperature, etc. His blood counts were confined to five cases of manic-depressive insanity with care that the technic be identical. The first case was a female of nineteen with the maniacal form. On admission had hallucinations of hearing, consciousness clouded, disoriented as to time and place, memory defective, rambling in her conversation; no well defined delusions, great motor excitement with tendency to impulsive acts. No disease insight. Temperature always normal. Fifteen counts were made. The next case was a male of

thirty with the maniacal form, consciousness clouded, flight of ideas, etc., yet oriented, memory unimpaired. Six counts were made. The third case was a mixed form in a female of forty-seven. Consciousness somewhat clouded, some retardation of thought and action; no delusions, no interest in her environment. Twenty-two counts were made. Case four was the maniacal form in a female of thirty-three. She had hallucinations of hearing, consciousness clear, oriented as to time, place and persons, no memory defect, judgment biased by delusions, no disease insight, had little interest in her surroundings. Forty-five blood counts made. The last case was of the maniacal form in a woman of fifty-seven. No hallucinations, consciousness clouded, mistook those about, completely disoriented, psychomotor activity marked. Only four counts made. The results of his examinations he summarizes in a table and draws the following conclusions from his observations: "1. There are no pathogenic blood changes in cases of maniacal phases of manic-depressive insanity. 2. Anemia is not a causative factor nor always an accompaniment of this psychosis. 3. The hemoglobin and red cells are frequently, if not always, increased during an attack of excitement. 4. A leucocytosis is an almost constant accompaniment and, I believe, a result of psychomotor activity. 5. That an increase in weight accompanies mental improvement."

2. *Hallucinatory Delirium of Acute Alcoholism.*—The statement of an acute alcoholic detailing the characteristic hallucinations of this disease with the delusions arising therefrom.

3. *The Progress of Psychiatry in* 1902.—Urkuhart mentions the paper of Dr. Warnock on Pellagrous Insanity and two papers by Dr. Mott, pathologist of the London County Council. The first deals with Stimulus in Repair and Decay of the Nervous System, setting forth the danger to neuropathics in the use of alcohol. He claims that stress is an important factor in the etiology of tabes. The second paper treats of syphilis as a cause of insanity. He states 70 to 80 per cent of paretics are found to have had syphilis and accepts the thesis: "No syphilis, no general paralysis." Dr. F. G. Crookshank's paper on "The Frequency, Causation, Prevention and Treatment of Phthisis Pulmonalis in Asylums for the Insane, as well as Dr. Eric France's paper on "The Necessity for Isolating the Phthisical Insane," has led to greater interest in this important subject, and the means for prevention urged are: early diagnosis, isolation, limitation of size of asylums, checking of overcrowding, improved ventilation, attention to dietary and sanitation in detail. Asylum Dysentery has been given careful study and is found not to essentially differ from ordinary dysentery. Night nursing has been given considerable study, with better care of patients at night. Marked advancement has been made in the matter of training nurses. The London County Council has established a Laboratory of Research at Claybury Asylum, where much attention has been given to clinical problems. Toxemia has been studied carefully and Dr. Ford Robertson claims all forms of insanity in normal persons is of toxic origin. An effort has been made, with some success, to treat incipient mental disorders in general hospitals. Some study has been given to asylum dietaries. Dr. Mercier's two books, a small Text-book on Insanity, the other a systematic treatise on Normal and Morbid Psychology, are notable books. The paper devotes itself solely to progress in England.

4. *Puerperal Insanity.*—The percentage of insanity due to pregnancy, parturition, the puerperal state and lactation, according to the report of the English Lunacy Commission, for five years, amounts to 6,4 in the private class and 8,1 in the poorer classes. At Claybury the statistics closely correspond to the above. Of 3500 admissions 259, or 7.4 per cent, suffered with insanity from the above specified causes, and are divided as follows: 56 from pregnancy or 21.62 per cent, 120 occurred during the puerperal state, or 46.33 per cent, and 83 were associated with lactation, 32.43 per

cent. The type of disease with pregnancy and lactation has nothing distinctive, while that of the puerperal state is marked by intense delirium with wildness and hallucinatory delusions, with permanent religious and erotic traits. Some cases present some mental disturbance to develop to insanity in the puerperal period, others again present mental symptoms during the puerperal state to be markedly alienated during lactation. Only 12 per cent of the cases were unmarried, yet of the insanity of pregnancy 25 per cent were unmarried, 8 per cent of the puerperal cases and only 3 per cent of the lactation cases. In the insanity of pregnancy it is mostly of the depressed type, while in the puerperal cases a maniacal type prevailed, and of the lactation cases the majority were of the depressed form. In 40 per cent of the cases of puerperal insanity symptoms appeared within the first two weeks and more than a third of these during the first week. Almost without exception the early symptom was loss of sleep followed by restlessness, distrust and suspicion, irritability followed by delirious excitement. Hallucinations of hearing were six times more frequent in these cases than of any other of the senses. Disorientation as to time and surroundings is common, as well as that she fails to recognize her own identity. The confusional state is followed by uncontrollable and restless violence, attended by profound physical exhaustion. The patient is often antagonistic to her husband, has erotic delusions, is immodest in conduct and language. Marked sexual excitement often exists. Suicidal thoughts are most common in the lactation cases occurring in nearly half, while in insanity of pregnancy it was noted in 41 per cent, in the post-puerperal in 21 per cent. The tendency to injure the child more common to the lactation cases. A previous record of hysteria is found in quite a percentage of these cases, but it is hardly sufficient to advise hysterical girls not to marry. Brown hair and gray eyes predominate. The age of greatest incidence was between twenty-five and twenty-nine for the insanity of pregnancy, and that of the puerperal period, while in the lactation cases from thirty to thirty-four. Heredity was present in 50 per cent and most so in the puerperal and pregnancy cases. This is considered the most favorable form of insanity, yet is very hazardous to the offspring. Insanity in early pregnancy is favorable and often recovers before confinement, while that of later gestation is apt to continue in an exaggerated degree and may become chronic. Most of the puerperal cases recover quickly, although permanent dementia may result. Cases of sudden onset show a slightly greater tendency to recover than those of gradual. Albuminuria, which is frequent, renders the prognosis grave. Gradual onset occurred in the cases becoming chronic and those dying. The death rate is the highest in the insanity of pregnancy and lowest in the puerperal cases. The pathology of these forms of insanity is not definitely established; some hold that various products of the changes incident to the processes of pregnancy and the puerperal state are the cause, in order words toxic, while in the lactational insanity the exhaustion often incident to this process may be the chief cause. The treatment must naturally depend on the fundamental condition. The induction of abortion in insanity of pregnancy has not proven beneficial. The patients need constant watching owing to their sudden outbursts of violence and suicidal ideas. Commitment to an asylum is generally best, unless the patient is so situated financially that she can have all the advantages of an institution provided in her own home. Puerperal cases should be treated at home if possible. The essential treatment is "compulsory superalimentation." Sulphonal is useful to induce sleep in motor excitement, paraldehyde is satisfactory, but not so good as a combination of chloral and bromide. A dull, listless condition may occur from which the patient is to be roused by a change in surroundings. Complications are to be treated according to their indications.

5. *The Care of the Insane.*—The last quarter of the nineteenth century

has witnessed wonderful strides in the care of the insane throughout the world. Until very recently the bars, locks, crib-beds and massive furniture were appalling, to say nothing of the devices for mechanical restraint. Of course this innovation in the treatment of a class considered so dangerous met with violent opposition. Exercise was confined to the "bull-yard," while now many patients are permitted almost unbounded liberty. Further occupation has been provided for most of the patients, instead of being compelled to spend their days on the ward benches, they are now helping in the shops, kitchen and farm work. Instead of the wards being bare of all ornamentation and hence cheerless and depressing, they are now comfortably and attractively furnished. The treatment of the insane today consists of "pleasant and sanitary surroundings, good nursing, proper medical attendance, suitable diet, entertainment and congenial occupation." One of the crying needs still is suitable buildings and appliances for the treatment of the acute cases, possibly recoverable under proper care, where association with chronic cases would be avoided, the appliances, both medical and surgical, provided for correcting and relieving physical ailments to which the mental disorder is often due. This would be a saving to the State in the long run by making the patient again productive, instead of possibly dependent for the balance of his life.

6. *Tent Life for the Insane.*—Dr. Wilsey cites in detail the benefit to a case treated in a tent nearly twenty years ago, and with marked and lasting improvement and recovery, and believes "the complete change in the whole existence of the patient was the important curative agents," and he warns us not to let means of diversion become monotonous, and those physicians who are the most versatile in providing for the patients' mental diversion will be the most successful.

7. *A Graded and Systematic Plan of Outdoor Exercise for the Demented Insane.*—The physical health demands the first attention in the amelioration of advanced dementia. A sclerotic state of the vascular system is largely responsible for the atrophic condition of the alimentary tract, resulting in more or less chronic states, which cause the defective metabolism. Consequently the dietary must be suited to this condition and therefore quality is more important than the quantity of food, and it must contain the largest possible amount of tissue-building material with the smallest possible amount of detritus. Digestive ferments and other medication may be employed to improve the patient's digestion. An improvement in the mental condition soon follows the betterment of the digestive process: the patient becomes more attentive, more tidy in his habits and more intelligent in his actions. The next essential is an abundance of exercise in the open air, and for this purpose the patient should begin with the simplest possible, one requiring little or no mental effort, and in time the patient will be found capable of performing more complicated work and even making himself useful in the shop. The games and occupation should be adapted as much as possible to the patient's individual liking. The faithful systematization and gradation of the amount and sort of out-door exercise has reduced the number of filthy cases to less than one-fifth of one per cent of the entire population of the hospital. The etiology and type of the disease must of course influence the possibility of either permanent or partial improvement. The most marked benefit has occurred in secondary dementia, particularly that following melancholia, while that following mania is less susceptible to instruction and suggestion. Dementia from organic brain disease is to be the least benefited. Great benefit is to be derived from this method of treatment in psychopathically inferior individuals and the allied forms of adolescent insanity, including hebephrenia and dementia precox. The results already attained warrant its further development and use.

8. *Extensive Injury to the Head.*—This case has been frequently re-

ported at various times from that of the receipt of the injury in 1857 until the patient's death within the past year. The first account embraces the details of the accident, which consisted of the tearing away of nearly the whole of the right side of the calvarium from the superciliary ridge to the occipital bone. At the time the patient suffered only slightly from shock, but no signs of cerebral concussion, faintness or nausea. The wound soon healed with no further symptoms than a dull headache. This report appeared in the *Buffalo Medical and Surgical Journal* for October, 1873, with the statement that the patient was in vigorous health at the time and only experienced a sense of fulness in the head when stooping or exerting himself. The next report of the case was by Gray, published in the *American Journal of Insanity,* April, 1876. This was followed by Dr. Bergtold's article in the *Medical Press of Western New York,* in 1888, written from his observation of the patient in the Buffalo General Hospital the year previous. In 1883 a slight unsteadiness of gait appeared which in 1888 had become a genuine paralysis, affecting the left extremities and right side of the face. Memory intact, no aphasia or headache, yet occasional attacks of dyspnea of asthmatic character; no cystic paralysis, yet some loss of control of the rectal sphincter. Sleep and appetite good. Deficiency of bone on right side of head with a depression of 5 x 6 inches. Special senses normal, speech slow and labored from paralysis. Mastication and swallowing interfered with, tongue protruded to left, lower lip pendulous. Slight atrophy of trunk and extremities, left arm nearly powerless, spastic pronation of hand and flexion of fingers, spastic contraction of left leg which was dragged in walking. Cutaneous sensibility intact; muscles respond to galvanic and faradic currents. No delusions or hallucinations, but patient irritable and emotional. Dr. Bergtold drew the following conclusions: "There probably began immediately subsequent to the injury an atrophy of the cerebral tissues situated beneath the site of the right parietal bone; this atrophy included the motor centers for the left arm and leg and the cortical centers for the facial and hypoglossal nerves; the atrophy, being slow, the remaining right cortex took on vicariously the duties of these atrophying centers; there gradually occurred (within the past five years) degenerative changes in these right compensating parts and further compensation did not take place in the left hemisphere." Thirteen years later he was admitted to the Buffalo State Hospital, being quite helpless and his mental faculties more affected. He was childish and irritable with paroxysms of anger, when we would try to strike. He could use his right arm, but both legs were firmly flexed at the knee and the right leg could only be moved feebly. Left arm spastic and joints contracted. Knee-jerks exaggerated, especially on the right. No paralysis of right side of face, tongue and lips paretic, eyelids drooping, tongue tremulous and atrophied, but protruded in the middle line without deviation; swallowing interfered with, speech almost unintelligible, drooling of saliva constant. Marked external strabismus, left eye noticeably protruding. Pupils unequal, sluggish in reaction to light and accommodation, vision imperfect, hearing about normal. Sensation apparently intact. Memory not seriously impaired, appreciative and tidy. No convulsions or attacks of unconsciousness. Died two years after admission. Autopsy showed a large deficiency of bone of the skull, where the scalp was adherent to the dura, which was thickened. Whole right hemisphere flattened and atrophied, but no evidence of injury or loss of brain tissue. Convolutions noticeably smaller than on left, of a shrunken, atrophied appearance, sulci gapping. No atheroma of the blood vessels. After hardening the brain was sectioned according to Hamilton's method, when a marked difference in the size of the hemispheres is shown. The most striking feature was a considerable area of degeneration of the subcortical white matter with numerous cavities just beneath the cortex and the hollowing out of certain convolutions. Slight softening of white matter through whole right hemisphere, yet no breaking down. Cavities confined to individual convolutions and not connected or noticeable externally except by slight depression. No other marked changes. The examination after staining showed a diffuse fibroid thickening and degeneration of the walls of the blood vessels in both hemispheres, but most marked in the right, the lumen being occasionally occluded. The vessels here showed a decided infiltration of round cells. The vessels on the right fuller than on the left. But few of the large pyramidal cells left and these shrunken and degenerated. A general increase of the neuroglia elements throughout the tissue. On the left side there is a slight degeneration of the tangential fibers in the cortex with projection fibers preserved; on the right tangential fibers absent at the top of the convolutions, yet seen at the sides. Degeneration of both motor and sensory tracts in the medulla and cord, most marked in those from the right hemisphere. These conditions in the cord explain the exaggerated reflexes, ataxia, spastic rigidity and gradual progress of the paralysis. The increased atmospheric pressure on the brain had diminished the capacity of the lymph canals and blood vessels, thus inducing the general atrophy from lack of nutrition, and this being progressive explains the character of the paralysis. The paper is accompanied by several photographs of the patient and the brain sections.

9. *Therapeutic Notes.—Cascara.*—Dr. Dewey extols the preparation known as "Kasagra," it being palatable and efficient. He advises that it be given in a dram before breakfast followed by a glass of hot water, and one or two drams followed by water at bedtime. It has proven effectual in the great majority of cases, even in those where constipation is a habit. Serum treatment of morphine intoxication. The researches of L. Hirschlaff are reported, who prepared a serum from rabbits by giving them morphine in increasing doses, which he found efficacious in opium poisoning and morphine addiction. Use of iron of animal origin. Hemoglobin may be given successfully in anemia and chlorosis. Mental disturbances due to hypnotism. Prof. Mendel's report is: "Careful distinction must be made between the cure of an affection and the alleviation of its symptoms by hypnotism. No physician claims that it is possible to cure with it an organic affection susceptible of demonstration with our modern diagnostic measures. But hypnotic or waking suggestion is undoubtedly capable of banishing, usually temporarily, but sometimes permanently, the most widely diverse symptoms of the affection, but without curing it. Success is dependent in this case, as in all cases of suggestion, o nthe greater or less skill of the suggestor, his personality, on external circumstances and on the degree of receptivity of suggestion by the patient. A number of cases are known, in which mental disturbances developed in predisposed individuals under hypnotization. Some of these cases occurred with charlatans, but in others the hypnosis had been conducted by physicians." Arteriosclerosis. The causes are cited and the treatment outlined, of which intestinal antisepsis is an important factor. The diet advisable is given in detail.

W. A. McCorn (Patterson).

## MISCELLANY

Two Cases of Sarcoma of the Spinal Cord. Senator (Prakt. Vratch., No. 24, 1903).

Tumors of the cord, until recently varieties inaccessible to treatment, have of late excited considerable interest. The author presents two highly instructive cases. In one case, that of a woman, sixty-nine years of age, there was a history of ten months' duration, that began with a sensation of formication in the toes and pains in the feet; to this was later added inability to flex the knees, burning and pains in the soles, which spread upward to the hip joint. During the last six months patient lost all power of locomotion, suffered from convulsive twitchings in the lower extremity, at times so pronounced and painful that the knees were forcibly flexed over the chest. There was also retention of urine. Patient lies recumbent on the back and is unable to move the lower extremities. Passive movements also hardly possible. Knee reflexes very much exaggerated. Babinski's sign present. Percussion of the muscles calls out painful twitchings. Stroking of the soles or legs is attended with clonic contractions of the lower extremities and of the abdominal muscles. Abdominal reflexes absent. Sensation lowered in the lower extremities. This condition grew gradually worse until the sensation of pain and temperature entirely disappeared, while that of touch disappeared over a still greater area, extending upward. Late in the disease there occurred curvature of the spine at the third lumbar vertebra. In the course of the disease the body of the patient grew, as it were, shorter, the chest approached the pelvis. The abdominal muscles were almost in a continual state of contraction. Dyspnea and attacks of tightness in the chest, Cheyne-Stoke's respiration and marked slowness of the breath (eight per minute) supervened. Lumbar puncture during the early period of the disease brought forth 10 to 12 cc. of a limpid liquid, with but little albumin. After the second puncture 5 cc. of one-half per cent solution of socaine was introduced into the subdural space with remarkable improvement to the contractures and pains, which lasted for three hours. Of the drugs employed, scopolamine (hyoscyamine) seems to have given the best results. The autopsy revealed a tumor of the dura mater, which was microscopically seen to be a sarcoma. The second patient was admitted to the hospital with pains in the back, inability to move the legs, and complaining of tormenting thirst. The trouble began seven months before with pains in the back, between the scapulæ, which would at times become so severe as to render her unable to work. Some four weeks before admission, the soles began to swell; this was followed by swelling of the abdomen, while two weeks ago patient became unable to move the left leg. There is no retention of either urine or feces. Patient recumbent on the back, indifferent, consciousness slightly impaired. Knee reflexes exaggerated, Babinski's sign present, ankle clonus indistinct. It is rather difficut to pick up the patient, as the spinal column is quite stiff. Patient complains especially of pains in the legs. Autopsy (patient died on the third day of admission to the hospital) gave the following: The heart slightly hypertrophied, lungs somewhat adherent, the thyroid gland hypertrophied, full of cysts; the bladder distended, its mucous membrane covered with necrotic spots; ovaries thickened. The vessels of the brain markedly calcified; the spinal column much congested; in the region of the third and fourth thoracic vertebræ, to the left, there is seen a hard swelling, which is microscopically a fibro-sarcoma. The interesting feature of this case is the very rapid course of the disease. The first stage, what the author calls the stage of neuralgia, was quite short, while the stage of paraplegia, that usually occupies months and years, was hardly extended over two weeks.

ROVINSKY (New York).

CASE OF TUMOR OF THE SPINAL CORD, WITH NECROPSY. Sinclair Gillis and Flashman (Australasian Medical Gazette, Nov. 20, 1902).

Diagnosis based on history of numbness in left hand (seven months' duration), followed by weakness in left hand and arm, and later by weakness in the right arm and in legs. Marked analgesia, anesthesia slight, except in left hand; anesthetic and analgesic areas being dissociated, thus, apparently indicating lesion of central gray matter in upper cervical region of cord. The existence of neuralgia, to a certain extent, in the left occipital region, tactile sense but slightly involved, indicated, possibly, syringomyelia. Subsequently there was great spread of analgesia (slight involvement of tactile sense), wasting of muscles of right palm, rigidity and weakness of right arm, followed by same condition in the left, spastic paralysis of lower extremities, involvement of sphincters, absence of pain, and, finally, death by bulbar paralysis. Postmortem: Brain and meninges normal. Attached to the spinal dura mater, on the right side, and a little posteriorly, about a quarter of an inch below the foramen magnum, was a growth the size of a shelled walnut. It was irregularly bossed, and attached to the dura by a narrow pedicle, and lay free in the subarachnoid space. It protruded through the foramen magnum one half inch, lying behind and to the right of the cord, which it compressed in the region of the first and second cervical segments. Cord here somewhat softened. A horizontal section close below the foramen magnum bisected the growth, leaving the lower fragment attached a quarter of an inch below to the dura mater, and the upper by a fine filament, to the medulla. Cord not invaded by growth, and, beyond softening in region of growth, microscopically normal, section of growth showed it to be a sarcoma, round celled variety. Nerve cells, all regions of spinal cord normal (Nissl method). Few scattered degenerated fibers in the ventrolateral white columns, especially ventral portion (Marchi method). Nearly whole medulla exhibited extensive fiber degeneration, probably due to pressure. Both inferior cerebellar peduncles showed well marked degeneration, affecting one-third of fibers, due to pressure on direct cerebellar tracts and external arcuate fibers. Pyramidal tracts slightly involved. Degeneration up into pons, limited to region of mesial fillet.

J. E. CLARK, New York.

ARTERIO-SCLEROSIS AS A CAUSE OF NERVOUS DISEASE. M. Allen Starr (Medical Record, July 4, 1903).

A careful study of two hundred consecutive cases of apoplexy in private practice has shown that in 80 per cent of these cases there have been distinct prodromata of the apoplectic attack. There were various symptoms which have usually been regarded as neurasthenic, pointing to a disturbance of function in the brain, chiefly in the cortical activity. Dulness and hebetude, difficulty of clear thinking or of remembering events of recent occurrence, or a general sense of perplexity. Others complain of a temporary sensation of numbness in one limb or one side of the body, or pains, or visual or auditory symptoms. Such symptoms are to be traced to malnutrition of the neurones, resulting from arterial disease. The writer advises thorough investigation of the condition of the blood vessels, especially the heart, and tension of the arteries, and when the condition of arterial sclerosis is recognized, appropriate treatment will make much of the so-called neurasthenia of middle age disappear.

The majority of the diseases of the spinal cord are equally traceable to disease in the vessel wall. Anterior poliomyelitis, bulbar paralysis, and ophthalmoplegia, when not distinctly infectious, are due to rupture or thrombosis of some branch of the anterior spinal artery supplying the anterior gray horns of the spinal cord. Myelitis, whether disseminated or transverse, if not due to infection, is due to a rupture of a blood vessel which has suffered from disease. Spastic paralysis, commonly known as Erb's syphilitic paraplegia, is now recognized as due to malnutrition of the dorsal region of the spinal cord, with consequent descending degeneration of the lateral columns. The cause of the malnutrition is syphilitic endarteritis obliterating the spinal blood vessels. Senile paraplegia, in which a gradually advancing weakness culminates in a slowly progressive paralysis of the legs, with imperfect control of the rectum, is due to obliteration of the blood vessels, to the thickening of their walls, and to a consequent malnutrition of their parts. Combined sclerosis is due to the same cause. Disease of the peripheral nerves is occasionally due to endarteritis of the small vessels. The various forms of neurasthenia and neuralgia are frequently due to the same cause. Treatment by iodide of potassium is frequently indicated for a time in high tension pulse. The writer believes thyroid extract is of use in some cases of temporary high tension pulse, and has noted especial results in cases of migraine, where the tension is usually high. W. B. Noyes, New York.

MENINGITIS WITHOUT ANATOMICAL LESIONS. J. Birnbaum (Münchener medicinische Wochenschrift, July 21, 1903).

It occasionally happens that patients die very suddenly with all the symptoms of a severe meningitis and yet only a slight exudate and perhaps a moderate degree of swelling and hyperemia of the brain is found post mortem. The history of the case reported by the author is unique in that the patient was ill for fifteen weeks, and that absolutely nothing was found post mortem in the meninges. Bacteriological examination, however, revealed the presence of a *meningococcus*, and the cerebrospinal fluid withdrawn during life showed a slight coagulum. The case was looked upon as one of meningeal sepsis without anatomical lesion. The cause was probably infection from a gum-boil which had been incised with extraction of the diseased tooth, two days before the acute onset of the illness.

JELLIFFE.

PROCEEDINGS OF THE ANNUAL SESSION OF THE GERMAN ASSOCIATION FOR PSYCHIATRY, AT JENA, April 20, 21, 1903.

The following critical summaries and papers were read and discussed. A critical summary upon the employment of isolation in the treatment of cases of mental disease, by Mercklin; papers upon the anatomical cortical region, by Vogt; upon the cytohistological localization of the human cortex, by Brodmann. A second critical summary upon the pronouncement of the Prussian Minister of Justice on the 9th of October, 1902, concerning the conduct of experts in the determination of irresponsibility, was read by Thomsen. A report upon the activity of the statistical commission named in the past year, was read by Hoche and Aschaffenburg, and a paper on the contribution to the knowledge of cretinism was read by Weygandt. Wollenberg demonstrated preparations of brain tumors. Liepmann read a paper upon the course of ideas. The third critical summary upon the idea and the significance of dementia, was read by Tucsek. Papers were read upon the construction of a sanitarium for nervous patients at the public expense, in Rasenmuhle, in Göttingen, by Cramer on the experimental studies with reference to the pathogenesis of acute psychosis, by Berger; upon the significance of special medication in the treatment of imbeciles, by Lacqer, and Binswanger demonstrated three cases of postsyphilitic dementia. JOSEPH SAILER, Philadelphia.

TABES AND THE MARITAL RELATION. A. Pitrés (Journal de Médecin de Bordeaux, July 12, 1903).

The author here presents a statistical article based on a study of 240 tabetics and dealing with the influence of tabes upon fecundity and the health of the offspring in marriages in which one parent was afflicted with locomotor ataxia. The 31 celibates of this number are dismissed by the author without comment. The issue of the remaining 209 amounted to 483 children; of whom 197 were still-born or died in the early months, and 286 survived; making an average of 2.31 births to each couple, with but 1.36 living children. Carrying the analysis further, the author states that of the 209 marriages, 42, or 20 per cent, were absolutely sterile, 32, or 15 per cent, resulted in still-births or death shortly after birth; while in the remaining 135, or 60 per cent, there were living children in the relatively high proportion of 2.11 per family. The high mortality seen in the offspring of tabetics, the author believes, is attributable to the antecedent infection rather than indirectly to tabes; and this infection is, in the majority of cases, syphilitic. He cites figures which go to show that the mortality of children born before development of tabes in either parent was 44 per cent.; while those born after the appearance of tabes showed a mortality of but 28 per cent. This finding furnishes, in his opinion, additional proof of the statement made by Régis, to the effect that the offspring of tabetics have a better chance of life and health when born in the later stages of the parents' disease than at a period nearer to the antecedent infection. As to the outcome of his investigations looking to the determination of the influence of syphilis upon the mortality of tabetics' children, the following figures are quoted: Of 77 male subjects who had undoubtedly had syphilis, 187 children were born, of whom 33 per cent were still-born or died in early infancy, and 67 per cent survived. Of 34 who denied antecedent syphilis, 81 children were born, of whom 20 per cent were still-born or died shortly after birth, and 80 per cent survived. With few exceptions, Pitrés finds that the surviving children of tabetics are uninfluenced by the parents' disease; their physical and intellectual development proceeding normally as that of other children; and of the 286 children of tabetic parents, whom he kept under observation, many were alive at twenty years, and some had attained thirty and thirty-five years at the time of writing. In none did tabes or Friedreich's disease develop, neither was there evidence of any other nervous affection. None showed signs of physical or mental degeneracy, nor was the influence of syphilis apparent in any. On the other hand, some developed diseases independent of all hereditary influence; such as tuberculosis, rheumatic cardiopathies, etc. The great majority were, however, healthy in body and mind.

JELLIFFE.

CHOREA IN PREGNANCY. Cecil Wall (Brit. Jour. of Obstet., June, 1903).

It has often been observed that the movements of a choreic patient are closely akin to those normally employed in the expression of emotions. In a young child, before the highest centers have developed coordinating control, such movements are alone present; it is only by a process of education that spontaneous movements of early infancy become coordinated, and thus capable of purposive action. Rheumatism is undoubtedly, in the majority of cases, associated in some way with the chorea of childhood. So, too, during pregnancy, rheumatism plays an important part in the etiology of chorea. Buist found a personal history of rheumatism in 45 out of the 226 cases that he analyzed. The author's statistics show that at least 16 of 37 patients had previously suffered from some form of rheumatism. Twelve more had had chorea in childhood without other rheumatic manifestations. Antecedent chorea may suggest a rheumatic taint, but cannot be taken as conclusive evidence. In pregnancy, defective mental development seems to vie with rheumatism and previous chorea as one of the predisposing factors, rendering patients liable to an attack of chorea. The determining cause for chorea is not infrequently to be found in worry, for which the pregnancy is the chief cause; in case the patient is worried by the knowledge that her child is illegitimate. Fear of dystocia may be another inciting factor. Chorea in pregnancy seems to be determined by mental display, overstrain and shock. Secondly, the determining cause is only effectual when it acts upon a brain whose power of control is somewhat lowered by the pregnant state, and in addition is unstable in consequence of (1) antecedent chorea; (2) antecedent rheumatism or a similar debilitating condition; (3) a defect in development.

JELLIFFE.

# Book Reviews.

A DICTIONARY OF MEDICAL SCIENCE. By ROBLEY DUNGLISON, M.D., LL.D. Twenty-third Edition. Edited by Thos. L. Stedman, A.M., M.D. Lea Brothers & Co., Philadelphia and New York.

Dunglison's Dictionary has always occupied the first rank among the medical dictionaries of this country, and in its successive editions it has been brought more or less up-to-date. With the present edition, the 23d, it has undergone a much more radical revision than has ever before taken place in its history, bringing it once more into the foreground as a serious and trustworthy dictionary.

Notwithstanding the immense number of new terms which have been added, the work has been kept within reasonable limits and the editor is to be congratulated on his careful work in the field of cross-reference and in his elimination of much waste matter. We can congratulate both editor and publisher alike on the successful accomplishment of this twenty-third edition. R. BROWN (New York).

Mikroskopisch-topographischer Atlas des Menschlichen Zentralnervensystems mit beuleitendem Texte von Dr. Otto Marburg (Ehemaligen Assiatiulin au Neurologischen Institute der Weiner Universität) Mit Eineun Gleitnost von Prof. Dr. H. Obsteiner. Mit 5 Abbildungan un Texte und 30 Tafeln nach Originalin des Akademischen Malers A. Kiss. Franz Deuticke, Leipzig and Wein, 1904.

This work embraces the author's well known systematic scheme of study of the central nervous system. His experience is shown in that this work is concise, lucid and practical. The Atlas is particularly timely as clinical neurology of today demands an accurate working knowledge of nervous anatomy. American colleges are just inaugurating this work. The Atlas consists of 125 pages of descriptive text illustrated by five text figures and thirty full-page plate drawings. The latter are especially beautiful and clear. The sections are stained after Weigert-Pall and Czokor carmine. The serial and natural order in which the spinal cord, medulla pons, brain stems and brain are considered, renders it easy for the student to gain the essential and practical facts. Sections in three planes aid much in orientation of the important anatomical structures. An introduction by Prof. Obersteiner accompanies the work. We can heartily recommend this working atlas of the central nervous system to every thorough-going student of neurology. L. PIERCE CLARK.

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**Removed to 64 W. 56th Street, New York**

Vol. 30 December, 1903 No. 12

# The Journal of Nervous and Mental Disease

The Official Organ of

The American Neurological Association
The New York Neurological Society
The Philadelphia Neurological Society and
The Chicago Neurological Society

MANAGING EDITOR AND PUBLISHER
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64 W. 56th Street, New York City

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S. Karger, Karlstrasse 15, Berlin, Germany. H. K. Lewis, 136 Gower Street, London, W. C. E Steiger & Co., 25 Park Place, New York. Gustave E. Stechert, 9 E. 16th Street, New York.

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Printed by the Alliance Press Company, 38 W. 18th Street, N. Y. City

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ISSUED MONTHLY $5.00 PER YEAR

FOREIGN SUBSCRIPTION, $5.60 PER YEAR

Entered June 20th, as second-class matter, Post Office at New York, N. Y., Act of Congress, March 3, 1879

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## Original Articles.

### ENORMOUS TUMOR OF THE POSTERO-PARIETAL REGION, WEIGHING OVER HALF A POUND; ABSENCE OF LOCALIZING SYMPTOMS UNTIL LATE IN THE HISTORY OF THE CASE; OPERATION; DEATH.

*History of the case by Dr. Dercum.*

By F. X. Dercum, M.D., and W. W. Keen, M.D.

The following case, because of the extreme difficulty of localizing the growth, of the nature of some of the symptoms present, and because of the enormous size of the tumor, is of unusual interest and importance.

H. W., age twenty-six, single, formerly civil engineer, later hotel proprietor. Examined April 23, 1902.

Family History. Mother died at the age of thirty-six with pulmonary tuberculosis. Father living and in good health. Patient has one sister, who is also living and well.

Personal History. Was healthy at birth. At the age of one year, suffered from a discharge from the right ear and subsequently lost his hearing upon this side to a very decided degree. An examination made some years later revealed a perforation of the drum. At long intervals, discharge from this ear recurred. During the past year or two the ear has again been a source of annoyance, as it has again been discharging a thick offensive fluid.

The patient had the ordinary diseases of childhood, from which he made good recoveries. He has had, since childhood, no serious illness, that is nothing that confined him to bed. He had at various times occasion to consult physicians for indigestion and occasional attacks of headache. He had been educated as a civil engineer, and some years ago, while working in some mines in Kentucky, had received at various times quite severe blows on the top of his head, but not severe enough to cause loss of consciousness. At another time his legs were seriously bruised by an accident.

For a year past he has been manager of a hotel at a seaside resort, and in March, 1902, his hotel was suddenly placed in danger from fire. At this time he exerted himself very greatly in order to secure the safe exit from the hotel of his guests and other persons, and also to save their trunks and valuables. Although he had been placed under sudden and serious strain, he betrayed no special symptoms save some headache. This headache persisted, and he believed that it was in some way connected with his eyes. He thereupon consulted Dr. G. E. de Schweinitz. Dr. de Schweinitz reports that he found at this time no changes whatever in the eye-ground, nor any defect of any of the ocular muscles. About one month later, on April 23, the patient again visited Dr. de Schweinitz, and upon this second examination a double optic neuritis was observed. It was most marked in the left eye, the field of which was somewhat contracted. There was also paralysis of the right external rectus and a consequent diplopia.

On April 23 he was first examined by myself. Questioned again as to his past history, he now recalled that for a number of months past he had noticed a little uncertainty of movement in the right arm, that he noticed occasionally that he would make a mistake in the act of picking up his pen, that is, that his fingers would not grasp the pen but would go a little to either side of it. He distinctly recalled that upon one occasion when about to help himself to butter he put his fingers instead of his knife into the butter. These acts of slight loss of control of the movements of the right arm were not, however, frequent. He also noticed awkwardness of movement in his legs, in trying to go upstairs two steps at a time. He added also that he had recently had several attacks of vomiting, especially in the early morning, before he had taken any food.

Present Condition. Complains of a dull headache. Asked to localize it, he states that it is more marked over the left eye. At times the headache is diffuse over the entire forehead. He has no headache over the parietal region, over the vertex or over the occiput. At times he has a feeling of tension about the head and at times also he is dizzy. Occasionally, if he stoops to pick up something from the floor, he is obliged to get up slowly; otherwise the pain in his head grows worse. Has no ringing in the ears. Has not vomited for several weeks past, but not infrequently feels inclined to vomit.

Physical Examination. Gait normal. Station normal. States, however, that he has a feeling of uncertainty while making the Romberg test. Stands fairly well upon either leg alone. There is no intention tremor. Tongue protruded in the median line. The tongue is slightly tremulous. There is also a little tremor and unsteadiness of the lips. The left eyelid droops slightly. The right nasolabial fold is possibly a little shallower than the left. There is no inequality of the mouth. There is marked deafness of the right ear, which is due evidently to an old otitis media. The grip of the right hand is 84; the grip of the left hand is 74. There is no change in the handwriting, though the patient himself declares that it is somewhat shaky. There is no ataxia. Both knee-jerks are minus, but are well reinforced. There is no ankle clonus. The Achilles jerk is present and apparenty normal. There is no Babinski sign. There are no sensory losses, though the patient complains of a feeling of numbness upon the inner aspect of the lower portion of the right thigh and upon the inner aspect of the right calf. There is loss of stereognostic perception.

His pulse is 70 and his temperature is 98.2.

Urine Report. Amber; specific gravity, 1,025; reaction acid; no albumin; no sugar; urea, 1.6 per cent.

Microscopic Report. Negative.

Blood Report. Erythrocytes, 4,650,000; leucocytes, 10,200; hemoglobin, 70 per cent.

Because of the history of long-standing disease of the right ear, and because of a possible brain abscess, it was thought wise to subject the ear to thorough surgical exploration. Dr. Charles K. Mills, who saw the case in consultation at this time, fully agreed with me as to the advisability of this procedure.

Dr. de Schweinitz now made another eye examination, and reported as follows:

Mr. W. has a paresis of the right external rectus. He has double optic neuritis with many hemorrhages of the succulent and inflammatory type. Beyond this the examination revealed nothing new.

On May 12, 1902, the patient was operated upon by Dr. J. Chalmers da Costa.

A two-inch semilunar incision was made over the right mastoid, all structures being divided to the bone. The cartilaginous aural canal was then separated from the bony wall, and the mastoid chiseled. This was found decidely hard and completely ossified; the opening was enlarged with rongeurs, and the middle and posterior fossæ of skull were exposed and explored. The dura and brain tissue were found apparently normal, and hence the dura was not opened; the parts were thoroughly cleaned, hemorrhage controlled by torsion and ligature. A small strand of iodoform gauze was inserted, the aponeurosis was closed with catgut and the skin with silkworm gut. The drainage was removed at the expiration of forty-eight hours.

The patient made an uneventful recovery from the operation, and for a time was better, being free from headache, vertigo or vomiting. On May 20, however, in the morning, he complained of some vertigo and nausea. About noon had his wound dressed; suffered from nausea. Induced vomiting by means of his finger. Felt some relief after this vomiting, though the nausea persisted more or less for the remainder of the day. In the evening complained of a slight headache.

On May 25, 1902, he was re-examined by Dr. Mills and myself.

Station. A slight increase of sway.

Right knee-jerk slightly diminished; left knee-jerk slightly diminished.

Right ankle clonus slight disappearing; left ankle clonus, zero.

Right plantar reflex, zero; left plantar reflex, normal.

Faint hypesthesia of the inner aspect of lower portion of right thigh and right leg.

June 1, 1902. Symptoms as before. Tested for astereognosis upon the soles of the feet, it was found that he was somewhat uncertain as to the position of a pencil when the latter was placed in various positions upon the sole of the right foot. In one instance he clearly mistook the direction for transverse when it was longitudinal. Upon the sole of the left foot he made no errors whatever. Further, he did not recognize little balls of paper placed between the toes of the right foot as readily as when they were placed between the toes of the left foot.

June 3, 1902. Re-examination by Dr. Mills and myself.

Right knee-jerk, minus; left knee-jerk, minus.

Right ankle clonus, slight disappearing; left ankle clonus, zero.

Right plantar reflex, sometimes absence of response; left plantar reflex, normal. Other symptoms as before.

June 7, 1902. This evening (about 7.30) complained of nausea and vertigo. This attack appeared rather suddenly and was quickly followed by an attack of vomiting. Vomiting was not followed by any relief, though it recurred at intervals; it was accompanied by considerable retching. The vomited material consisted of a thick viscid mucus and some bile. Nausea and vertigo had almost disappeared by the following morning, after patient had slept fairly well.

June 10, 1902. Examined by Dr. W. G. Spiller. Present, Dr. Mills and myself.

Left knee-jerk, minus; right knee-jerk, more diminished.

Sensation for touch preserved in each lower limb and appears to be equal upon the two sides. The same is true of pain sensation. If hypesthesia is present upon the right side, it is exceedingly slight.

Movement of the toes to irritation of the sole of the right foot is that of flexion of all the toes, except the great toe; the great toe does not move. Upon the left side the movement is that of flexion, including that of the great toe. The only difference between the two sides is that the great toe of the right side does not move at all.

Left ankle clonus, zero; right ankle clonus, zero.

The Achilles jerk is prompt upon the left side, while upon the right side, tapping the tendo-Achillis causes repeated contractions, like ankle clonus.

Patellar clonus is not present upon either side.

Resistance to passive movements seems to be fully normal in each limb.

He stands erect without swaying, even when eyes are closed.

The gait is not peculiar.

Hemiasynergy is not present in either lower limb when the patient is reclining.

The grasp of each hand is good and apparently normal.

The biceps jerk, triceps jerk and wrist reflexes are not very distinct upon either side.

Sensation to touch and pain in the upper limbs is equal upon the two sides, and is normal. States that his right hand is numb, that this is so distinct that he is certain it exists and yet upon testing it cannot be determined.

The patient states that since the operation, or possibly before the operation, he has been a little weak in the right lower limb, and also in the fingers of the right hand, arm and right leg; he complains of being awkward with the right hand. There is apparently some disturbance of sense of position. On attempting to put the first finger of the right hand on the first finger of the left hand, or vice versa, when eyes are closed, he has much difficulty in touching the desired objects. He is able to tell correctly any movement of his fingers or toes when his eyes are closed. Stereognostic perception is normal in both upper limbs.

Hemianopsia is not present. The pupils are equal. There is no nystagmus in looking to either side. Movement of the facial muscles is normal upon either side. Movements of the muscles of mastication are normal upon either side. There is no word-deafness, no word-blindness, and no symptom of sensory or motor aphasia. His memory is not quite as good as formerly. Says that he has become more irritable than formerly. Also that within the last twelve months he is not as good at figures as formerly; he cannot add or multiply as well as formerly, and this change he regards as very perceptible, and is even greater than the failing of memory. He has more difficulty than he formerly had in understanding the meaning of what he reads.

The patient is not as able to tell whether a pencil is placed lengthwise or crosswise upon the sole of his right foot; he makes no error upon the sole of the left foot.

June 20, 1902. Re-examined by Dr. Mills, Dr. Spiller and myself.

Lower limbs are well developed, although he says he is not as stout as a few years years ago, weighing about twenty pounds less at the present time.

Resistance to passive movements of the thigh and leg, of each side, is normal and equal on the two sides.

Sensation to touch and pain in the lower limbs normal, though possibly tactile sensation is slightly diminished in the right lower limb as compared with the left. This diminution, if present, is slight. There is a slight diminution in the right lower limb to pain, tactile and temperature senses.

The patellar reflex on the right side is possibly slightly diminished; on the left side a little more pronounced than on the right side. Ankle clonus not obtained on either side. Plantar irritation upon the right side produces a slight movement of all toes in flexion, except the great toe, this not being moved at all; movement of the other toes is slight. On the left side, the movement of all the toes is that of flexion.

The Achilles jerk is present on each side and is about normal.

The gastrocnemius reflex is present on each side. These reflexes, Achilles and gastrocnemius, being equal on both sides.

Station erect is good with eyes closed. Gait normal. Stands well on each foot alone. Resistance to passive movement in the right upper limb is normal; the same in the left upper limb.

Sensation to touch, pain and temperature is normal in the two upper limbs. There is possibly a slight diminution in the right arm, but this is questionable.

Biceps jerk, triceps jerk and wrist reflexes are about normal upon the two sides. The sense of the position of the fingers and toes of each side seems to be normal, but he is not always certain of the right great toe. He seems to be able to tell correctly movements of the left great toe. Apparently there is no loss of the sense of position in the upper limbs, or at least the loss is not pronounced. The movement of the right upper limb, when the eyes are closed, would suggest a slight loss of the sense of position.

The stereognostic sense is well preserved in both hands, although he occasionally makes a mistake in trying to differentiate between a dime and a cent with the right hand. These mistakes are infrequent. He never, however, makes any mistake with the left hand. Upon the sole of the right foot, there is again noted, as at previous examination, a tendency to make errors as to the position of the pencil or penknife when placed upon the sole of the right foot. Upon the sole of the left foot no errors are made at any time.

The patient was re-examined July 5 and July 29 and presented no noticeable change of symptoms. On September 5, 1902, he was re-examined by me. His symptoms were the same as at previous examinations, save that there was present an area of undoubted tenderness over the skull in the left post-parietal region. This area in front begins at eight and a quarter inches from the glabella and about half an inch from the sagittal suture. It extends as far back as to within one and three-quarter inches of the occipital protuberance. It is about two and three-quarter inches in its longitudinal diameter. It extends downward over the parietal region for about two inches. It appears to be oval in shape. The patch is sensitive upon superficial pressure and made worse upon deep pressure, and pain is also readily elicited by friction. The patient states that this area has been more or less noticeable since August 31.

Of late the patient has suffered but little from headache, having had but one severe attack since he was last examined. His diplopia is about the same. Dr. de Schweinitz reports his vision as perhaps slightly better. There is little or no change in the paresis of the left external rectus. There is less swelling of the optic discs than there was.

Because of the new symptom of tenderness in the post-parietal region, I submitted the patient to another examination on September 11, at which Drs. Mills and Spiller were also present. Strange to say this symptom of tenderness had at this examination almost or completely disappeared, so that it could not be confirmed by either of my colleagues. Otherwise the symptoms were as noted at previous examinations. It was thought by Drs. Mills and Spiller that possibly there was a slight awkwardness in the movement of the fingers of the right hand and of the toes of the right foot. Difference in the movement between the digits of the two sides of the body, however, was so slight as to be open to discussion. This remarkable symptom of tenderness, both superficial and deep, upon the postparietal region never subsequently recurred, although new symptoms made their appearance, while some of the old ones gradually became more pronounced.

On October 17, 1902, he again presented himself for an examination, and it was now found that in addition to the symptoms which he had presented before, there was a slight hypesthesia of the left side of the face. It was most marked over the middle distribution of the trigeminal and to a less extent externally over the superior. Mr. W. was able to detect all tactile impressions directly, but hypesthesia was undeniably present. He also described a subjective sensation of numbness and a crawling sensation in the left side of the face; also that the left side of the face felt somewhat stiff. The slight difference in the nasolabial folds, noticed in the early history of his case, was still present, but had not become accentuated. There was no involvement of the muscles of mastication. The patient had had very little headache, and only a slight attack of nausea some two weeks ago. About this time his case was studied by Dr. William Osler, who, with myself, thought that this numbness of the face suggested a deep basic lesion upon the left side. Dr. William M. Sweet now made several careful skiagraphs of the head, but no shadows were revealed that seemed in any way to suggest the location of a tumor. After a consultation with Drs. Mills and Spiller, it was determined to advise an exploratory operation of the base upon the left side. The patient's symptoms were at this time gradually becoming more pronounced, although the general symptoms were as before. The optic neuritis was now of high grade, with beginning degeneration of the optic nerve fibers, with resulting depreciation of vision (de Schweinitz). The fields were contracted, but there was no hemianopsia, the pupillary reactions were unchanged. There was a palsy of the right external rectus, thirty degrees, and a palsy of the right superior rectus (possibly inferior oblique) of eleven degrees. There was some widening of the palpebral fissures, giving the appearance seen in hydrocephalus.

On November 3, 1902, Dr. Keen made an osteoplastic flap in the left temporal region, but beyond determining a distinct resistance posteriorly, the operation resulted negatively as regards localization of the growth. Mr. W. made a perfect recovery from the operation and subsequently returned to his home. He was not seen again until December 17, 1902, when it was found that the hypesthesia of the left side of the face had disappeared. There was, however, a slight, but unmistakable hypesthesia present in the lower limb and to a less extent of the right upper limb. The plantar reflex was not obtainable upon either side. The knee-jerks presented the same difference as at previous examinations, as did also the ankle clonus. The grip of the right hand was 65; that of the left hand, 60. The patient stood well upon either leg alone. The tongue was protruded in the median line. There was no facial inequality. The atrophic changes in the optic nerve were somewhat more marked than at the previous examination. When tested for astereognosis, occasionally made errors with the right hand. No word-blindness. No word-deafness.

The patient now passed from under my observation. I did not again see him until March 18, 1903. Dr. T. Percival Gerson, of Lansdowne, under whose care he was, reported that he had gradually become hemiplegic, there being a very gradual loss of power making its appearance in the right side of the body, right leg and right arm, also that there had been very decided loss of sensation upon the right side and awkwardness of movement.

March 18, 1903. Examination by Dr. Dercum, together with Dr. Gerson.

Mr. W. presented the following symptoms: He entered the room with a decided hemiplegic gait, dragging the right leg and allowing the right arm to hang at his side. He was not able to stand upon the right leg alone, while the loss of power in the right arm was also very marked. There was a decided hypesthesia of the right arm, right leg, right side of the face and head and right half of the trunk. This hypesthesia was not sharply demarcated by the middle line. It was most marked in the distal portions of the extremities and became less marked as the middle line of the trunk was reached. The area of but slightly impaired sensation extended from two to four inches from the middle line to the right. The right knee-jerk was much exaggerated. The left knee-jerk was about normal. There was now persistent ankle clonus upon the right side. The elbow jerk was present upon the right side, but not pronounced. There was a slight von Bechterew reflex upon the right side.

The Babinski reflex was not present upon either side. Upon the left side the toes moved normally, but on the right side there was a slight movement of the four outer toes in flexion, while the great toe did not move at all. Right-sided homonymous hemianopsia was now present, although both visual fields were distinctly contracted. This hemianopsia could readily be demonstrated and was quite well defined by the middle line. Wernicke's symptom was not present. Astereognosis was now complete. There was also typical word-blindness. There was no word-deafness. The patient had, however, a distinct difficulty in the use of substantives. He was unable frequently to give the name of common objects, such as a book, even though he examined the book both by vision and touch. The symptom of anomia was not complete, but it was undoubtedly present. At this examination, tenderness in the post-parietal region was again sought for, but not elicited.

Because of the new features presented by the case, I decided to again advise an operation, and submitted the question to my colleagues, Drs. Mills and Spiller, who concurred in its advisability. Accordingly, on March 26, he was operated upon by Dr. Keen, who made a large exposure of the parietal region. The details of the operation are given by himself. The bone proved to be excessively vascular, and the operation had to be completed in three stages, the final result being the removal of an enormous sarcoma, weighing 264 grams. The mass was encapsulated and was removed without much difficulty, but the patient succumbed to shock and operation shortly afterward.

Although no autopsy was obtained, the fact that the tumor was encapsulated makes it exceedingly probable that only one sarcomatous mass was present in the brain, and that all of the symptoms were referable to this growth. The interest of this case lies in the extreme difficulty of the localization and in the indefinite and often misleading character of the symptoms. It is remarkable that cortical disturbances, such as unmistakable astereognosis and alexia on the one hand and hemianopsia upon the other, were not present earlier in a case of tumor in this situation. The inference is justified that the growth originated subcortically and only late in the course of the affection produced cortical phenomena by upward growth and hemianopsia and basal phenomena by downward growth. In reviewing the symptoms we are impressed by the fact of the insignificant value of such a sign as a paralysis of one abducens or of a trifacial hypesthesia. In the present instance they were undoubtedly due to pressure from a distance, and as is well known, they frequently so occur from such cause. They are of themselves of little or no localizing value. However, the case does demonstrate the localizing value of astereognosis and slight muscular incoordination. In reviewing the history of the case, it will be noted that faint, but unmistakable astereognostic signs were present upon the sole of the right foot, and somewhat later upon the right hand, as were also faint, though somewhat inconstant symptoms of incoordination of the right fingers and right toes. Lastly, the symptom of local tenderness and pain in the post-parietal region, which was present for a few days only, assumes a vast importance. It was so fugitive that it was not present at the examination made a week later by Drs. Mills and Spiller, and was apparently absent at the examination made by Dr. Osler, and yet that there was abundant cause for such a pain, the finding at the operation of a large exostosis of the internal table in this very situation most conclusively proves.

To repeat, the history of the case and the very gradual evolution of localizing phenomena justify the inference that the tumor began subcortically in the post-parietal region, and that it grew downward and upward very gradually. It would seem to me that it would be a perfectly justifiable expedient and far preferable to what was done by ourselves in this case, when symptoms of one-sided trouble, vague in character, make their appearance, not to attempt an exploration of the base, which is always a matter of extreme difficulty, but to make a free osteoplastic exposure of the most probable area upon the lateral aspect. The free osteoplastic exposure is attended with little risk, and the exploration possible under the circumstances is, as a rule, very thorough. Had such a plan been attempted by us I am quite sure that the tumor would have been found, though early in the case it would doubtless have been found to be situated beneath the cortex. The lesson to be drawn from this case is to make free osteoplastic exposures in all cases yielding symptoms referable to one side of the cortex, no matter how slight these symptoms may appear to be.

The tumor apparently grew very rapidly in the last few months of the patient's life. The size and weight that it attained were truly enormous, and showed how very much pressure the brain can withstand without destroying the life of the patient.

### *Surgical Report by Dr. W. W. Keen.*

I first saw Mr. White in consultation with Dr. Dercum on October 29, 1902. The reasons for operating at the point selected, and also for the site of the prior operation which Dr. DaCosta made on the right side in the spring of 1902, have already been given by Dr. Dercum.

Operation November 3, 1902. I made a flap in the left temporal region (Fig. 1) practically the same as that for the Gasserian ganglion, including chiseling the zygoma so as to get down as low as possible. First, with a gouge I made a small opening in the bone, and then with the rongeur forceps enlarged it till it was 4 to 5 cm. in diameter. The bone seemed to be unusually thick, two or three times the ordinary thickness in this position. Exposure of the dura showed that it was very resistant and bulged considerably. As soon as it was opened the brain protruded. I attempted to lift the temporo-sphenoidal lobe with a broad spatula, so as to gain access to the pons, where the tumor, it was thought, might possibly be. It was, however, evident that the protrusion of the brain was such that I should do very extensive damage to the brain tissue if I lifted it to the desired extent. The cortex did not seem harder than usual, though the sense of resistance in the brain as a whole was quite marked.

In order to determine whether there was any possible distension by fluid, I punctured the lateral ventricle, but did not find any. The diminution of resistance when the ventricle was entered was very perceptible. I then incised the temporo-sphenoidal lobe to a depth of 3.5 cm. and introduced my finger into the incision. The white substance was normal to the eye. To the finger there was a distinct increase of resistance to pressure *posteriorly* as compared with the other three directions. After making several attempts to lift the temporo-sphenoidal lobe, and failing to see even as far as the site of the Gasserian ganglion, I abandoned the operation. He made a perfectly smooth recovery, the highest temperature being 99.6 degrees.

Second operation, March 26, 1903. First Stage. Owing to the development of new symptoms, especially hemianopsia, after a consultation with Drs. Dercum, Mills and Spiller, we decided that it would be wise to do a second operation. The site of the tumor was now more accurately located in the parietal lobe extending forward perhaps as far as the fissure of Rolando and well backward into the occipital lobe. It will be observed that when at the first operation I introduced my finger into the substance of the temporo-sphenoidal lobe, I recorded at that time that the resistance posteriorly was distinctly greater than in the other three directions. This also would confirm this diagnosis as to the situation of the tumor.

I outlined a flap, the anterior border of which crossed the fissure of Rolando about at its middle, and passed nearly vertically upward, 9 cm. long (Fig. 1). Parallel with the median line, but 2.5 cm. away from it, I made an incision 13 cm. long; this reached into the occipital lobe. The posterior incision was

12 cm. long, as indicated in the diagram. The hemorrhage from the scalp was exceedingly profuse. At every point where the bone was exposed sufficiently to chisel it, wherever a little vessel penetrated the skull, the blood poured out from the bone in a fountain nearly 1 cm. in height. These were stopped by Horsley's wax, the vessels in the scalp being clamped. I then proceeded to chisel the bone. Of course the incision was not all made at first, but in three sections, and the chiseling of one was completed before the next was begun. The bone also I found very vascular. Some of the veins of the diploë poured out distinct streams of blood. These also were stopped by Horsley's wax, but in spite of this a large amount of blood was lost. I could not understand why, especially at the posterior portion of the median incision, I did not get through the bone. While I was operating, one of my assistants, Dr. Craig, opened a vein in the right arm and infused 20 ounces of salt solution, to which were added 20 drops of 1-1000 adrenalin solution. Finally, as the patient had lost so much blood, we decided that the vascularity of the parts showed that the tumor lay underneath the proposed flap, that it was a very vascular sarcoma, and that if I continued the operation there would be so large a loss of blood

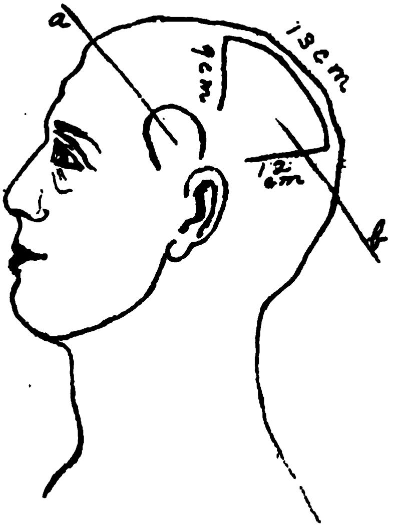

Fig. 1.

when I turned back the osteoplastic flap that it would probably cost him his life. I decided, therefore, to do the operation in two stages.

After the operation his temperature, up to the 29th, rose to a little above 100°, but from the 29th to the 31st fluctuated between 101° and 102.6°. In spite of the high temperature, he did not seem to be very sick, though his pulse was from 100 to 120.

Second stage. On March 31 we decided to reopen the skull and operate on the tumor. As I anticipated, a very severe hemorrhage from the tumor, I first clamped the common carotid with Crile's clamp and obliterated the caliber of the artery. After reopening the wound in the scalp, which was attended with very free bleeding again, I chiseled some places in the bone where I had not chiseled entirely through, especially in the portion parallel with the median line just posterior to its middle, and at last was able to turn back the flap. As soon as it was reflected, the difficulty of chiseling through the bone was explained. On the inner surface, just at the median edge of the flap and 3 cm. from its posterior end was a bony tumor, conical in shape, 2.5 cm. in height and 2.5 cm. in diameter. The chisel had traversed this bony tumor, where the bone was about two to three times its ordinary thickness. With the rongeur forceps I removed the entire bony growth. The dura was very soft, and at one point there was a deep depression corresponding to the bony growth. The tumor was evidently partly cystic. I then opened the dura corresponding to the osteoplastic flap. A considerable amount, estimated to be seven and one-half ounces, of bloody serum, so bloody that at first I thought it to be pure blood, escaped. This led me to ligate the common carotid and remove the clamp.

As soon as the brain was exposed, the entire opening in the bone, large as it was, was filled with a very large bulging sarcoma. It was so large and his condition was so critical, in spite of another infusion of a pint and a half of salt solution with 30 drops of adrenalin 1:1000 solution, that my judgment was against any attempt to remove the entire tumor, but I scooped out with my fingers a large handful. The hemorrhage was free, but not alarming. Packing with iodoform gauze, douching with hot salt solution checked the bleeding, and I closed the wound after gnawing away a part of the edge of the bony flap in order to allow the end of the gauze packing to protrude. I also ordered five minim doses of the adrenalin every four hours hypodermatically.

On the day after this operation his temperature rose to 103.2° and continued between 100° and 102° for nine days. It then fluctuated between the normal and 100°; meantime the wound had healed without incident. The packing was removed on the second day without any noticeable hemorrhage; the wound in the neck healed by first intention.

On April 1, the day after the operation, he moved his right arm once, but then was completely paralyzed on the right side for a week. Movement then began both in the right arm and leg, and he gained so rapidly that, after sitting up in bed for two or three days, I got him out on a chair, and on the seventeenth day he was able to walk down stairs into the garden of the hospital.

As his parents and he himself, appreciated the fact that if the tumor were not removed the end would be fatal, they consented to another and final attempt to remove the remainder of the growth. In view of his very good condition, Drs. Dercum, Mills, Spiller and myself, after a conference, determined upon the final attempt.

Third stage, April 21st. The flap was reopened very readily, the adhesions giving way under moderate leverage. The same peculiarity noticed before, was again evident, viz,—abundant hemorrhage not only from the scalp, but that every vessel which penetrated the bone became a little fountain. Again Horsley's putty stopped this quite effectually. After gnawing away the bone sufficiently and removing the diseased portion of the dura, I was able to reach the limits of the tumor. The tumor extended 2 cm. in front of the anterior border of my original opening in the bone, 1 cm. inferiorly and 1 cm. posteriorly. It extended in the middle line entirely to the falx. This large mass, which more than filled my entire hand, I was able to enucleate quite easily and cleanly. Fully one-half of the entire area of the falx was exposed as soon as the tumor was removed. The hemorrhage, especially from the middle and posterior portions of the wound was very profuse. Packing with iodoform gauze arrested it in part. Adrenalin was given together with a saline infusion continuously until 30 drops of 1 :1000 solution of adrenalin and a pint and a half of salt solution had been administered. No distinctly large vessels, which could be ligated were discoverable. Accordingly I had to trust entirely to packing and hot water. The flow was so profuse that adrenalin locally would have done no good. The wound was packed as firmly as was deemed advisable, the wound closed and the patient put to bed. He was in very profound shock and died about half an hour after being placed in bed. A culture from the surface of the tumor was given to Professor Coplin, who reported that the micrococcus pyogenes albus in pure culture was found. No abscess existed but the surface of the tumor was softened over a small area.

### REMARKS.

*Weight.* This tumor is the largest that I have ever removed from the brain. I have not compared it with any accurate list to see what others have exceeded it in weight, but so far as I know the only one which exceeds it, is that removed, with better success, I am glad to say, by Bramann. His tumor weighed 280 grms., 16 grms. more than this. The tumor here reported weighed 264 grms., more than half a pound.

*Hemorrhage.* In few, if any, cases have I seen more severe hemorrhage from both scalp and bone. This seemed to indicate that the tumor lay directly under the site of the final operations. In the bone, Horsley's wax answered admirably in arresting the bleeding. At the final operation the hemorrhage from the brain was so great that even quite firm gauze packing did but little good. No known large sinus, vein, or artery was opened, but the hemorrhage was universal and severe. The application of Crile's clamp and finally ligation of the common carotid on that side modified it but little.

*Exposure of the Falx Cerebri.* Only very rarely have I removed a tumor which exposed the falx. In this case the exposure covered at least one-half of the superficial area of the falx, though less than one-half of its length.

Dr. Spiller examined the tumor and made the following report:

"At the earlier operation by Dr. Keen, a large portion of the tumor was removed. The mass removed was easily broken, and had in the fresh state the appearance of a sarcoma. The portion removed was hardened partly in formaline and partly in Müller's fluid. After hardening had occurred the portion in formaline weighed 27 grms., that in Müller's fluid 49 grms. The part first removed has an irregular surface as though it had been torn away from a tumor mass.

"The part of the tumor removed at the later operation is much larger than that removed at the earlier operation, and two-thirds of its surface are sharply defined and nodular, as though it had been sharply separated in this portion from the surrounding brain tissue. Approximately one-third of the entire tumor, if one may judge from the form of this large tumor, must have been removed at the earlier operation. One side of the large tumor has a torn appearance, and from here probably the first piece of tumor was removed. The upper surface of the tumor is flat. The weight of the second portion removed, before any hardening fluid had been used, was 188 grms., so that the entire weight of the tumor, including hardened and unhardened tissue, was 264 grms.

"The tumor consists of small spindle-shaped cells without definite arrangement, and is not exceedingly vascular. It is a small spindle-cell sarcoma."

# STUDIES UPON THE CEREBRAL CORTEX IN THE NORMAL HUMAN BRAIN AND IN DEMENTIA PARALYTICA.

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(*Continued from page* 716.)

*Occipital Region.*—Coming to the posterior pole of the external cortex, the typical structure of the occipital lobe is represented by Plate VIII, Fig. 24, a section magnified 14 diameters from a block taken from the point indicated by the figure 8, Plate I, Fig. 1. The section is 6 2-3 microns in thickness, is 6 mm. in width at the widest portion, and 7 mm. in length, stained with methylene violet, fixed in Van Gehuchten's fluid, other technique as previously described. This convolution is seen to be quite different in shape from the two previously described of the temporal and parietal regions, having a broader base and narrower rounded vertex. The cortex is seen to be thickest on the lateral aspect *b,* thinner on the opposite lateral aspect *c,* and thinnest at the vertex in the vicinity of *a;* the central white medullary substance being reduced to a minimum. The outer layer varies in thickness, being thickest at *c.* Above this it is broken away for a short distance, is quite thin at the vertex, and becomes somewhat thicker on the lateral aspect *b.* Above *b* the section is seen to be cracked, an artefact resulting from its fixation upon the slide, also at *c* numerous small breaks are noticed, due to the microtome knife. Opposite *a,* and enclosed in ink lines, is the segment of this section, represented in Plate IX, Fig. 25, under a magnification of 100 diameters. The radial arrangement of the cells is well made out at the vertex, but not quite so readily seen on the lateral aspects of the gyrus here, but under a higher magnification can be distinctly made out. About the middle of the second layer, especially at the vertex, some very large pyramidal cells are to be seen singly or in groups, and approaching the Betz cells in type in some cases. The third or lower layer presents nothing unusual, and disappears below into the white medullary substance of the interior of the gyrus. Plate IX, Fig. 25, is a photomicrograph of the segment *a,* enclosed in ink lines in Plate VIII, Fig. 24, at a magnification of 100 diameters. Here the cortex is found thinner than in any of the preceding plates, measuring scarce 2 mm. in thickness. The first layer averages only .15 mm. in thickness, and in structure and arrangement of the cells is similar to this layer in the preceding plates. The second layer measures 1.15 mm. in thickness, containing small pyramidal cells above and quite closely packed together, but increasing in size below and being somewhat more scattered. About the middle of this layer a number of large cells are seen, many singly, but some arranged in groups in places.

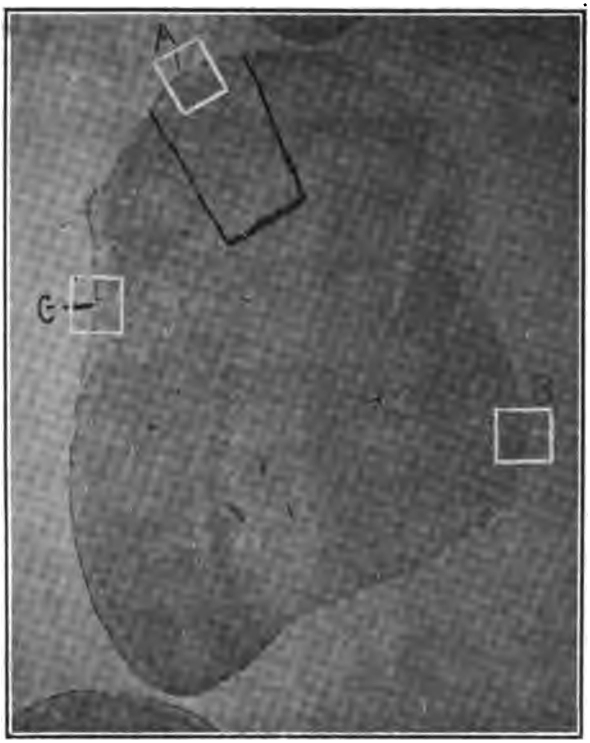

Plate VIII, Fig. 24.

Some of these are even larger than those found in the parietal convolution, and are intermediate in size between the Betz or giant pyramidal cells and the large pyramidal cells. These latter are not very numerous in this region, the small pyramidal cells making up by far the greatest part of the cellular elements of this layer. Below the middle of this layer is to be found a region, at *a*, almost exclusively made up of small pyramidal cells about .25 mm. in width. Between this and the lower border large pyramidal cells are again found intermingled with the smaller. The third or spindle cell layer is here .70 mm. in thickness, containing larger and smaller, spindle and irregularly polygonal cells, the lower border being gradually lost in the white medullary substance below. The nerve cells are found quite numerous and closely aggregated in this region, as counts by the same method and manner as in all previous sections, give here an average of 164 nerve cells to the square millimeter of surface of the section, and 199 neuroglia cells, the section being 6 2-3 microns in thickness and stained with methylene violet. The cells are thus seen to be more numerous than in any other region of the external surface of the hemisphere, with the exception of the posterior central convolution. The fact of the greater number of the pyramidal cells being small, permits of their closer aggregation, and no doubt is an important factor in the result of the nerve cell counts. The number of neuroglia cells averages higher than in any other region posterior to the fissure of Rolando, but they are less in number than in the anterior central convolution.

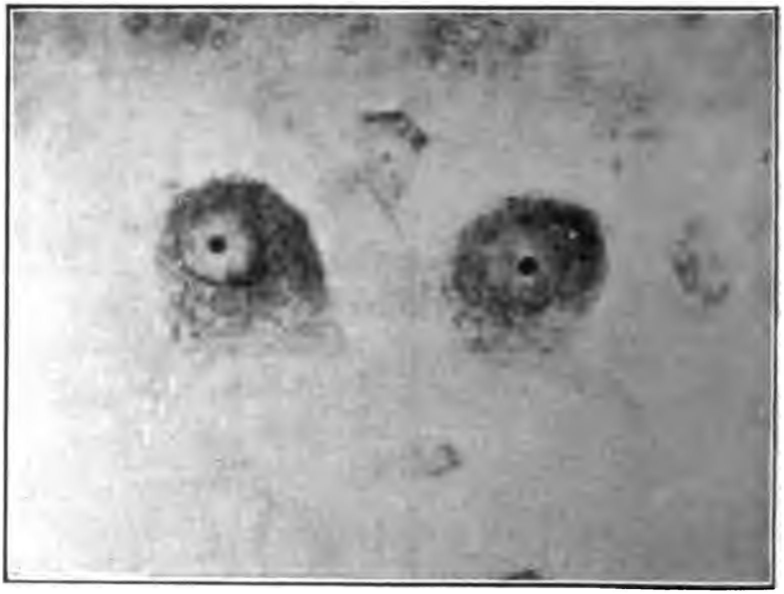

Plate IX, Fig. 27.

This includes the plates of the external surface of the cortex, but for the purpose of comparison, several plates will be introduced from other parts of the central nervous system. The first of these is a photomicrograph, Plate IX, Fig. 26, of several large pyramidal cells from the layer of large pyramidal cells of the hippocampus major or cornu ammonis; between the alveus, representing the white medullary substance of the ordinary gyrus, and the stratum radiatum containing the apical dendrite processes of these hippocampal pyramidal cells. This plate and the two following are not presented as a basis of comparison with similar regions of the pathological brain subsequently to be described, but to give an idea of the appearance of these cells in a normal brain as seen in a photomicograph of a Nissl preparation magnified 1,400 diameters, as distinct types in Nissl's classification.

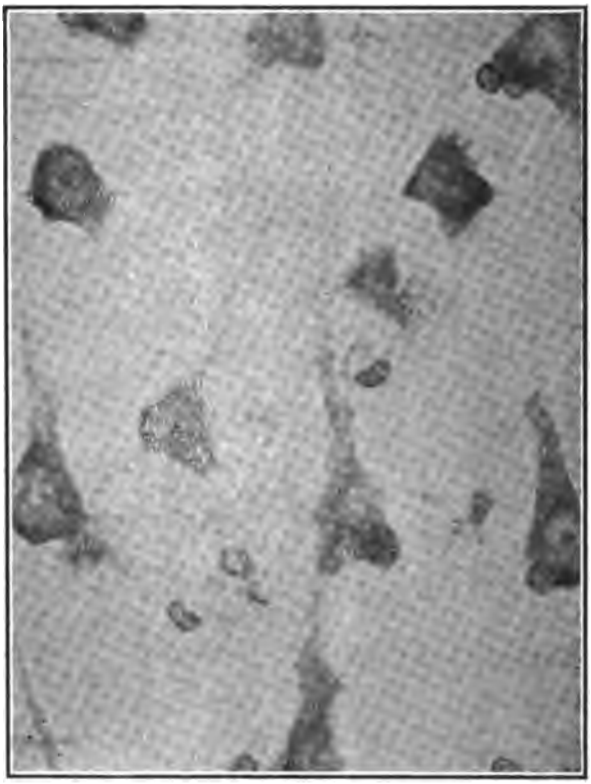

Plate IX, Fig. 26.

These large cells of the cornu ammonis are given as one of the four types of Group II of the stichochrome nerve cells in his classification; the other three types being (1) the nerve cells of the motor nuclei, (2) certain cells of the cerebral cortex, and (3) certain cells of the spinal ganglia. In shape these cells approach the larger pyramidal cells of the external surface of the cortex, second layer, but are somewhat more slender, of a narrow pyramidal shape, have fewer basal dendritic processes, and a long, somewhat slender, apical dendritic process. The chromatic substance here is made up of very small granules fusing into larger and smaller masses in places, so that in the photomicrograph it is difficult to differentiate and determine their arrangement accurately. In some of the cells, as in the cell to the extreme right of the plate, and partially out of the field, the rounded and linear granules above the nucleus, and extending into the apical process, are seen to have a general parallel arrangement. The nuclei of these cells are large, and contain a large, well marked nucleolus and a more or less irregular chromatic network. As this section was stained with methylene violet, numerous neuroglia cells are distinctly seen scattered among the pyramidal nerve cells.

From the cerebellar cortex a photomicrograph was taken of two Purkinje cells, magnified 1,400 diameters, and shown in Plate IX, Fig. 27. The section from which this photomicrograph was taken was situated on the external surface of the left hemisphere, cut transversely to the horizontal axis of the same, fixed in alcohol 95 per cent, stained with methylene blue: other technique similar to that of previously described plates. The section is 10 microns in thickness. These Purkinje cells were given by Nissl as typical examples of what he formerly described as Group II, arkyostichochrome nerve cells of the somatochrome class, in which was presented a striated appearance, with a network-like structure, united in a most intricate manner, thus having characteristics of both the arkychrome and the stichochrome cells. He now classifies them as one of the types of Group I, the arkyochrome nerve cells. In the Purkinje cell, to the right, the base of each of the two large dendritic processes is seen, one going off to the right, the other to the left, subsequently to divide and subdivide in the internal layer, not shown in the plate. The Purkinje cell to the left shows only one of these processes in this plane, that going off to the left which divides in the same manner as above described for the other cell. The large rounded nucleus, with its centrally placed nucleolus, is fairly well shown in the cell to the right, and contains a fine network of chromatic substance. The chromatic substance of the body of the cell is made up of larger and smaller irregularly-rounded chromophilic bodies, arranged in an indefinite network extending up into the base of the dendritic processes. A nuclear cap is seen above the nucleus of the Purkinje cell on the right. The closely-packed fine granular cells of the granular layer of the cerebellum are seen just below these two Purkinje cells, and are included in the class of cytochrome nerve cells in Nissl's classification. They contain a nucleus almost filling the cell body, and which is surrounded by only a very narrow rim of chromatic substance. Within the nucleus is a nucleolus, and in some of these nuclei several irregularly-arranged chromatic granules are also seen. Between the Purkinje cells is a small nerve cell with rounded body and an apical dendritic process. It contains a nucleus, nucleolus, and some chromophilic granules irregularly arranged in the cell-body.

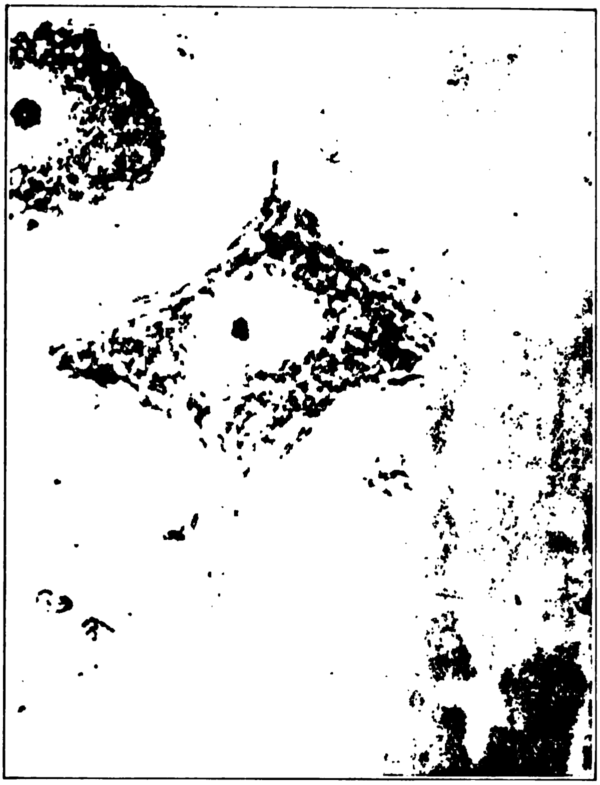

Plate X, Fig. 28.

The last photomicrograph in the series of the normal histological material is that shown in Plate X, Fig. 28, of a typical multipolar ganglion cell from the anterior horn of the lumbar enlargement of the spinal cord, under a magnification of 1,400 diameters. The segment of cord from which the section was taken was fixed in Van Gehuchten's fluid; this section is 6 2-3 microns in thickness and stained with methylene violet. This cell is, according to Nissl's classification, an arkyochrome nerve cell, Group I, of the somatochrome class, the chromophilic bodies being arranged more in the form of a network than in a parallel arrangement in the cell-body, though within the dendritic processes these bodies are more or less distinctly parallel in arrangement. The cell-body is irregularly polygonal in outline, with five large dendritic processes going off in different directions, and containing at the base, and for a considerable distance, linear-shaped chromophilic bodies, arranged in a more or less parallel manner and spreading out and connected with the cell-body network. This latter is made up of irregularly-rounded and elongated chromophilic bodies, arranged in an irregular network. The nucleus is large, rounded, slightly eccentric in situation, and contains a large rounded nucleolus and a pale indefinite network of chromatic substance. Numerous neuroglia cells are seen scattered about, singly or in groups, in the immediate vicinity of this cell. Part of another large ganglion cell is seen at the upper portion of the plate, on the left.

This concludes the very brief discussion of the cortex of the normal brain, in which an endeavor has been made to present typical sections of the main divisions of the external surface of the cerebral hemisphere by means of photomicrography, upon which the greatest care was employed, in order to give an accurate idea of the usual appearance to be seen under the microscope in the study of such sections in contradistinction to the common diagrammatic figures that one usually associates with such work, and which do not show the actual arrangement and structure, but rather the ideal or embodyment in one figure of what is to be found in parts of many. The writer, on the other hand, does not wish to be understood as not favoring diagrammatic or schematic figures in work of this kind, as he regards them as highly important and a valuable accessory to photomicrography and accurate drawings in the proper conception of the intricate structure and arrangement of the various portions of the nervous system as well as other systems. That there are minor variations from these plates in the different regions has been repeatedly stated, but the variation is only a minor one, and does not depart sufficiently from the type to be considered as more than a modification. Furthermore, in going over the various plates from the different regions it will be seen that there is a general uniformity of arrangement and structure of the cells, and that one region does not vary to any great extent from that of any other region. This is well illustrated by comparing the plates from the frontal region with those of the temporal region, in which it would be difficult to determine the one from the other. The Betz cells in parts of the motor cortex are a conspicuous modification, but when we consider that only a small part of the motor area contains these cells, and again that these cells, though large, represent but a minute fraction of the total number of cells in the cortex at this point it will be seen their presence makes but a slight modification in the general cortical arrangement as a whole. The pyramidal cells intermediate in size between the large pyramidal cells and the Betz cells are of especial significance to the writer as perhaps indicating that, with increased specialization of function, carried on through long periods of time, increased specialization of the structure and size of the cortical cells is going on hand in hand, and that perhaps the so-called Betz cells, now almost entirely limited to certain portions of the central convolutions, some time in the future may be found in all parts of the cortex. Their large size, with resulting large amount of protoplasm in the cell-body and large nucleus, the comparatively enormous amount of chromatic substance, as possibly stored up food products, all point to great capacity for storing up nervous energy to be given out when indicated as powerful efferent impulses, or, on the other hand, the capacity of receiving as equally powerful different impulses. Increased specialization in mental activity may result in greater numbers of nerve cells being set apart and increased in size and capacity for carrying out special functions.

*Paretic Brain.*—Turning now to Brain B, the case of dementia paralytica, from which all the succeeding plates were taken, we see in Plate X, Fig. 29, a photograph of the left hemisphere of the brain, natural size. The macroscopic appearance at first sight would seem to indicate that there was marked atrophy and shrinkage in parts of several convolutions, especially the anterior central and frontal convolutions; but a careful study of the sections taken from these regions shows this not to be altogether the case, or at least only to a slight degree. As any one who has studied brain topography to any extent well knows, there is much variation, not only in the shape and arrangement of any given convolution, but also in its size, so that great care should be observed in not mistaking such normal variation for atrophy. A study of the various plates shown here under a magnification of 14 diameters, containing the entire cross section from the several convolutions in the brain and comparing them to the preceding brain will make this point clear. Here and there a considerable quantity of the cortex has been torn away with the pia mater, whereas in many places small depressions are seen in which only a very small portion of the superficial layer of the cortex has been torn away. The convolutions are here well marked and the sulci very deep, so that there is a large area of cortical surface. The sulci in the middle portion of the hemisphere are widely open, owing to the support being placed only in the middle of the median surface of the hemisphere, so that the weight of the poles, anterior and posterior, caused these latter to become depressed, resulting in this conspicuous widening of the central sulci. The posterior pole of the brain—the occipital lobe—is practically normal macroscopically, and it will be seen later also microscopically, there being none or but very slight pathological changes in the cortex in that region. This photograph of the brain was taken before any blocks were removed, it having been previously fixed in 95 per cent alcohol. As each block was removed the exact location was noted and indicated in the photograph by the letters of the alphabet, and the place from which the block was taken enclosed in ink lines.

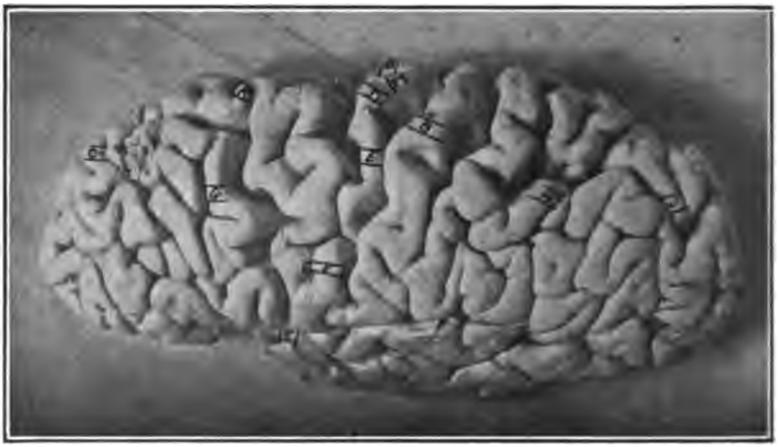

Plate X, Fig. 29.

*Frontal Convolution.*—This portion of the cortex is represented by the block K, taken from the external surface of the first frontal convolution in its middle third, the exact location being seen in Plate X, Fig. 29, K. The photomicrograph seen in Plate X, Fig. 30, was taken from a section 10 microns in thickness, and is magnified 14 diameters. The gyrus here is somewhat narrow, and the sulci on either side were quite open and deep, and from the macroscopic appearance one might at first be inclined to think that there was some atrophy of the gyrus; but a detailed study of sections from this region shows that this is not the case. Block K was fixed in 95 per cent alcohol; this section is 1 cm. in length, .63 cm. in width at its widest part, and, as above stated, 10 microns in thickness, being stained with methylene blue; the other technique being the same as for all the other plates. The shape of the section is somewhat that of a truncated pyramid, broader at the base and narrow at the rounded vertex. This mechanical arrangement makes the cortex thicker at the vertex than on the lateral aspects. At *a*, and enclosed in ink lines, is the segment represented in Plate XI, Fig. 31. The first or superficial layer is seen to vary somewhat in thickness, being thicker on the lateral surface below *b*, with another thickened portion below *a*, a third just to the left of the vertex, and finally at the lower part of the left lateral aspect. The second or pyramidal cell layer does not show quite as distinct arrangement of the cells in a radial direction as in the corresponding region of the normal cortex, and there is especially to be noted the numerous portions of minute capillaries of varying size and tortuosity, singly or

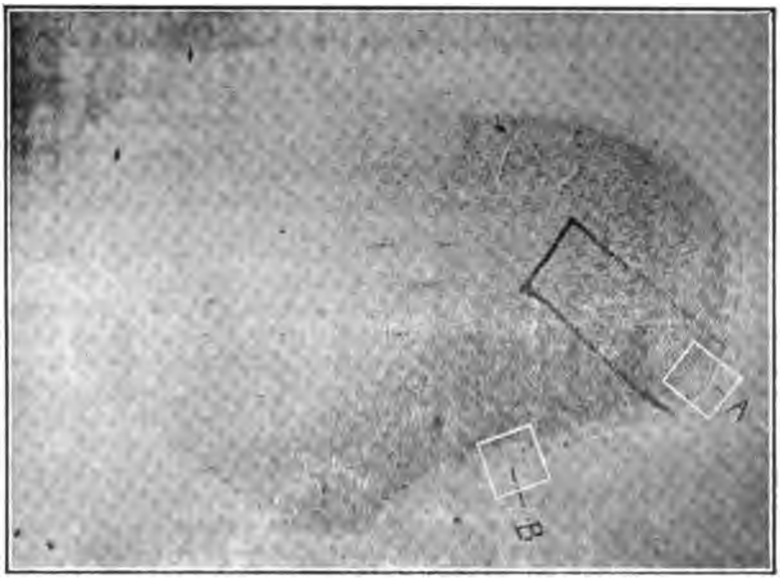

Plate X, Fig. 30.

branched. This second layer passes over into the indistinct third layer, which is lost below in the pyramidal-shaped white medullary center, which also contains numerous capillaries. Plate XI, Fig. 31, is a photomicrograph, magnified 100 diameters, of a segment corresponding in position to *a* of a section adjacent to and from the same block as that shown in Plate X, Fig. 30. The section is 6 2-3 microns in thickness, and of the same length and breadth as that shown in the preceding plate. Decolorization has been carried on to a considerable extent, about to the same degree as in Plate II, Fig. 3, and thus causing the plate to appear rather pale. Additional causes are the paucity of large pyramidal cells in the second layer, and the shrunken condition of many of the cells, as well as other pathological alterations. The upper layer averages .20 mm. in thickness, and contains for the most part scattered neuroglia cells. The second or pyramidal-cell layer is 1.80 mm. in thickness. Here is to be found a very similar arrangement of the cells to that in the corresponding region of the normal brain A. In the upper portion are to be seen the small pyramidal cells in considerable numbers, just below the superficial layer. Then they become more scattered, with a tendency to an arrangement into larger and smaller irregular groups, the cells becoming larger in the deeper portions. A little below the middle of this layer is found a narrow strip at *a*, where the small pyramids predominate almost to the entire exclusion of the larger ones, which latter, however, are seen above and below. Below this again are to be seen the larger pyramids, which finally give way to the irregular and spindle cells of the third layer. The nerve cells themselves show various changes. Many of them appear to be atrophied or shrunken, this process varying in degree in different cells. This in many cases has resulted in the formation of larger or smaller pericellular spaces as seen about many of these cells, as at P in this plate and in Fig. P of the text, for instance. Here a large pericellular space completely surrounds the cell. The basal dendrites are represented by only one small shrunken process given off at the left and extending but a short distance. The body of the cell is shrunken and irregular in contour. A large mass of yellowish pigment is seen at the base to the right. A small amount of finely granular chromophilic substance is found about and above the nucleus. The nucleus itself is displaced downward into the base of the cell-body, to the extreme left. It is small, irregular in outline, and contains a well defined nucleolus but no chromatic substance. The apical process is shrunken and irregular in direction, and contains traces of chromophilic substance in places. The chromatic substance in the large majority of these cells has become diffused and decreased in amount in variable degrees, so that a large number of the cells present the appearance of chromatolysis up to almost complete disappearance of chromatin in some of them. These latter present a pale, washed-out appearance, as shown by the cell marked Q in this plate and seen enlarged in Fig. Q, where only a small amount of chromatin is found in the apical process and a slight amount of pigment in the lower part of the base. Some cells show loss of chromatin only about the nucleus, and are spoken of as instances of central chromatolysis. Others have a disappearance of chromatin at the periphery of the cell-body only, and this condition is known as peripheral chromatolysis. The nuclei of many of these cells are found frequently displaced to one side, crowded into the base of a dendritic process, near the base of the cell-body, or near the base, or up into the proximal portion of the apical process. Many of these are irregular in shape and diminished in size. Quite a large proportion of the larger pyramidal cells contain light yellowish pigment in variable quantity and usually at the base of the cell, often extending into the base of a dendritic process. The cell P, above described, in addition to the pericellular space, demonstrates these last two points, the nucleus being crowded into the base of the dendritic process given off on the left and a considerable deposit of pigment being found at the base and extending into the right dendritic process. The cells containing this pigment are marked with a cross, adjacent to them, in this plate. It will be seen that they are to be found mostly in the middle and lower portion of this layer, and also some few scattered irregular-shaped cells in the third layer. The dendritic processes in many of these cells end abruptly beyond their base, and in but few cases can they be traced to any great distance from the cell-body. The apical process shares in this general atrophy, and often is curved and irregular in direction, instead of presenting a regular straight course towards the periphery of the cortex. In some cells the nucleus is difficult to distinguish, the limiting membrane being indeterminable and the nucleolus appearing to be in the midst and surrounded by only the cell-body, and usually in an eccentric position. Numerous blood vessels are here to be seen, all with thickened walls and pursuing a tortuous course. Large perivascular spaces are also seen about many of these blood vessels, as at *d*, for instance. The third, or spindle or irregular cell layer is here .90 mm. in thickness, and is made up of irregular and spindle cells, some of the former, as some of the cells of the second layer, contain pigmentary deposits. Here also the chromatin is found in diminished quantity, and various stages of chromatolysis are seen. The nucleus is diminished in size in many cases and does not appear to be as prominent a factor as in the normal cells of this region. The cells as a whole appear smaller in size and more or less atrophied, containing less protoplasm in the cell-body as well as a diminished amount of chromatin. The blood vessels are here also found to have thickened walls, and are tortuous in direction. At *e* is seen one of these vessels, with thickened walls and surrounded by a large perivascular space. The entire thickness of the cortex at this point is found to be 2.90 mm.; almost the same as in the corresponding region in Brain A, as seen in Plate II, Fig. 3. Counts were made of the nerve and neuroglia cells in various parts of this section in the same manner as in the previous sections, and in eight different fields of 36 sq. mm. each of the ocular net-micrometer from various portions of the two lower layers for the nerve cell counts, and in all three layers for the neuroglia cell counts. There was found to be an average of 100 nerve cells and 56.90 neuroglia cells to each square millimeter of surface of this section. The small number of neuroglia cells being due to the methylene blue stain employed and differentiation carried on to a considerable degree in a section 6 2-3 microns in thickness. In the section shown in Plate X, Fig. 30, 10 microns in thickness, and more deeply stained, the number of nerve cells is not quite so great, whereas the number of neuroglia cells is almost six times as great, although the two sections are quite near together and from the same block. This is due to the neuroglia cells in Plate XI, Fig. 31, being almost completely decolorized in the greater differentiation, whereas the cells, although paler, yet were not decolorized, and can all be made out. The same method of using the ocular net-micrometer was employed here as in all the other plates, and counts were made from eight different fields of thirty-six squares each from different parts of the second and third layers of this section, and the average number of nerve cells was found to be 82.91 to the square millimeter of surface of the section, as compared to 100 for the same area in Plate XI, Fig. 31, so that there are on an average less nerve cells in this section, 10 microns in thickness, than in the section from which Plate XI, Fig. 31 was taken, which was but 6 2-3 microns in thickness, the same stain being used in both cases. The nerve cells, however, are more deeply stained in this latter section, and appear more distinct, and there are also more large cells. The neuroglia cells in eight different fields of thirty-six squares each from various parts of the three cortical layers average 303 to the square millimeter of surface of the section, as contrasted to 56.90 to the square millimeter in Plate XI, Fig. 31. Here increased thickness makes a difference in favor of this plate, and in addition to that the deeper stain with less decolorization cause all the neuroglia cells to appear more prominent. As stated before it was found that sections from 6 2-3 to 10 microns in thickness contain all the nerve cells to be seen in one plane. As the neuroglia cells are much smaller in diameter and often arranged in dense clusters, there may be a greater number in a section 10 microns in thickness than in one 6 2-3 microns in thickness. (See Table II.)

*Central Region.*—Turning to the region posterior to the one just discussed, we come to the central region, or motor area, made up of the anterior and posterior central convolutions. Typical of the anterior central convolution in its upper portion is the section represented in Plate XI, Fig. 32, under a magnification of 14 diameters. The exact location of the block from which this section was taken is indicated by the letter D in Plate X, Fig. 29. The wide separation of the convolutions from one another, with resulting gaping sulci, is especially marked in this region. As previously mentioned, this is largely due to mechanical agencies, a central support permitting the opposite poles, owing to their weight, to become depressed and force open the sulci in this region during the process of hardening or fixation. In the removal of the pia mater, small portions of the adhering cortex have been removed in places. This is seen just below the letter O, and in many places in the convolutions both anterior and posterior to this. By referring back to Plate III, Fig. 7 of the normal brain

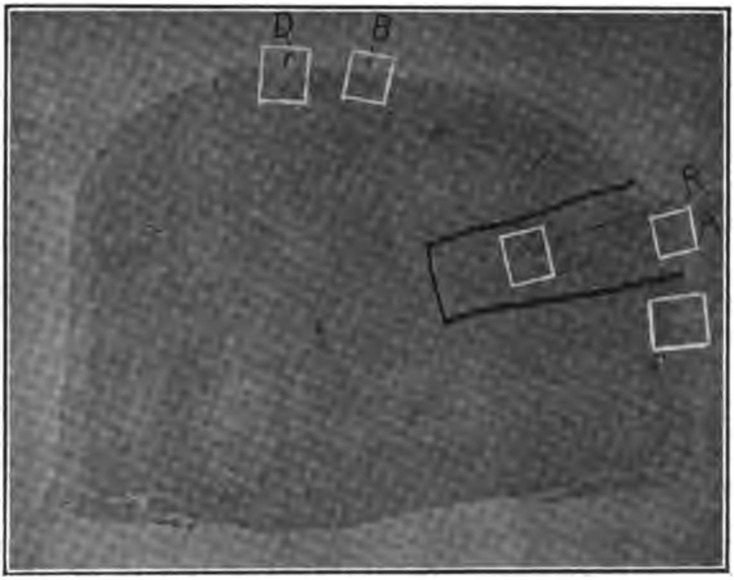

Plate XI, Fig. 32.

the right side of this cell. The general contour and lack of structure are seen in Fig. V. Here, too, neuroglia cells are seen lying upon and in close juxtaposition to the cell-body. Numerous blood vessels with thickened and tortuous walls and perivascular spaces are seen throughout this layer. The third or spindle or irregular cell layer here measures .90 mm. in thickness, and is made up of shrunken spindle and irregular cells, pale, and containing but a small amount of diffuse chromophilic substance. Many of them are surrounded by pericellular spaces of varying size. The letter W indicates one of these spindle cells seen in the drawing, Fig. W. The polar processes are here filamentous, and distorted in direction, the cell-body shrunken and containing but a small amount of diffuse chromatic substance, so that it is pale in color, the nucleus is small and indistinct, and contains a small nucleolus. The cell-body is partially surrounded by a distinct pericellular space, with a neuroglia cell upon the edge at one point. Numerous blood vessels with thickened walls and perivascular spaces are also seen in this layer. The entire depth of the cortex here is 3.05 mm., all three layers being slightly thicker than in Plate III, Fig. 8, of Brain A, the first layer being .05 mm., the second .15

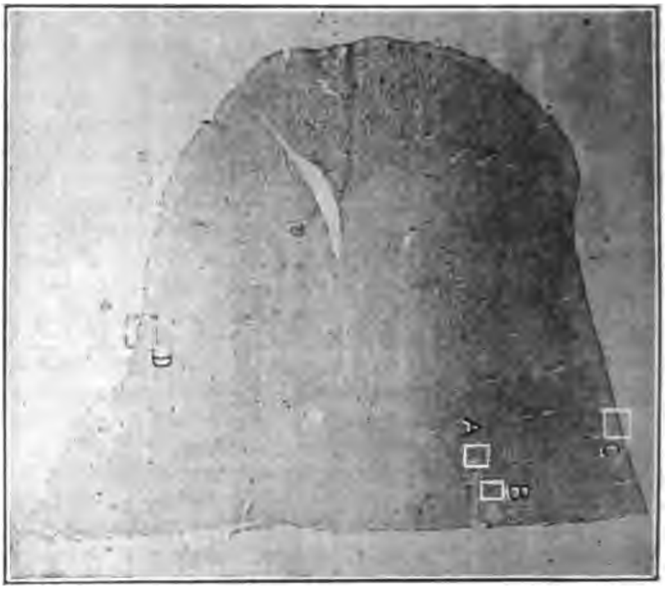

Plate XI, Fig. 34.

mm., and the third .20 mm., thicker than in the latter. As variations of this extent are found in sections of convolutions in close proximity, and even in different parts of the same section in any brain, it seems doubtful to the writer whether we can attach any special significance to such a slight variation, due possibly to mechanical causes, acting in embryonic or even post embryonic life. Counts of the nerve and neuroglia cells here show a decided falling off in number of the former. This section is 6 2-3 microns in thickness and stained with methylene violet. The same method with the ocular net-micrometer was used, counting the nerve cells in eight different fields of thirty-six square millimeters, each in various parts of the second and third cortical layers, and the neuroglia cells in the same number of different fields in all these cortical layers. The average number of nerve cells to each square millimeter of surface of the section was found to be but 39.60, whereas the average number of neuroglia cells to the same area was found to be 204. The number of nerve cells is thus seen to be much diminished as compared to Plate III, Fig. 8, the corresponding region of the normal brain A, where there are an aver-

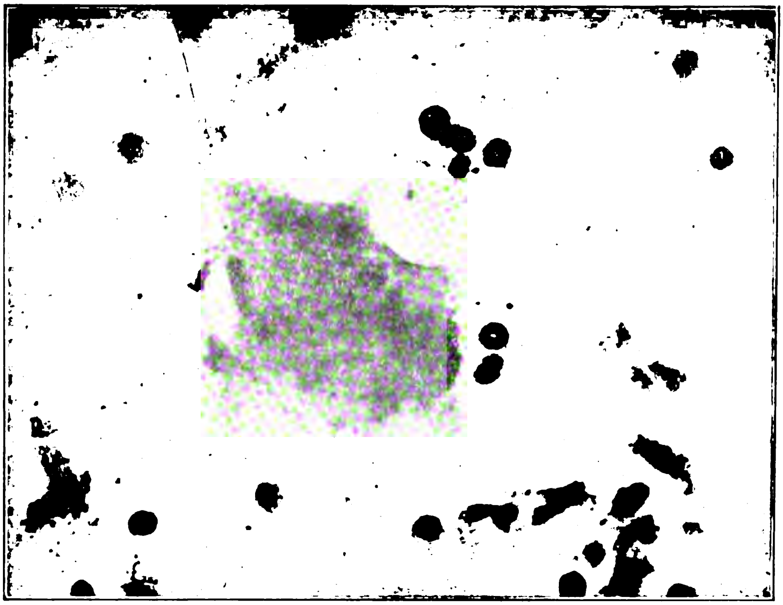
Plate XII, Fig. 35.

age of 95 nerve cells to the square millimeter of surface of the section. The number of neuroglia cells is also less here, being 204 in this plate in comparison to 293 in Plate III, Fig. 8. The latter section is, however, 10 microns in thickness, as compared to 6 2-3 microns for this plate, and as previously noted, owing to the small size and close aggregation of the neuroglia cells, more would be normally found in the thicker section, although this hardly accounts for the great difference, as both were stained in the same manner with methylene violet. Plate XI, Fig. 34, is a photomicrograph, magnified 14 diameters, from a section taken from the point L of the upper portion of the anterior central convolution of Brain B (see Plate X, Fig. 29), and as seen by referring to this latter, is from a more inferior portion of this gyrus

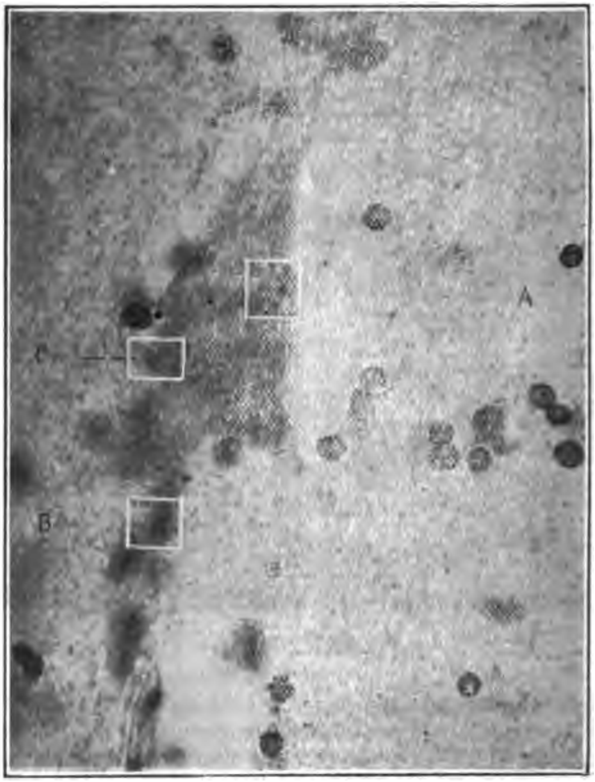

Plate XII, Fig. 36.

than the section represented in Plate XI, Fig. 32. This section was fixed in 95 per cent alcohol, is 6 2-3 microns in thickness, and stained with methylene violet. The first layer is seen to be fairly uniform in thickness at the lower two-thirds, as seen at *c*. Above, and at the vertex, and also along the anterior aspect of the convolution, as at *d*, the surface is more or less torn and irregular, due to carrying away of small fragments with the adherent pia. By referring to Plates XI, Fig. 32, and XIII, Fig. 39, the variations in shape and mechanical arrangement of the convolution in adjacent portions will be noted. In Plate XI, Fig. 32, the vertex is broadly rounded and somewhat flattened. In Plate XIII, Fig. 39, the vertex is more narrow and pointed, while in this plate, intermediate in position between the above two plates the vertex is less pointed. Large perivascular spaces are noted in many places throughout this section, especially marked in the upper portion to the left of the vertex, where but a fragment of the blood vessel is to be seen only at the lowest point of an enormous perivascular space. The radial arrangement of the cells is but poorly shown, the Betz cells seem to have disappeared almost entirely in the second layer, in marked contrast to the plates of the same magnification of Brain A. (Plates III, Fig. 7; IV, Fig. 9, and IV, Fig. 11.) Under a high magnification, however, the remnants of many of these Betz cells can be made out, showing various pathological changes. The other nerve cells also show a wealth of pathological change of varying degree and kind to which the nerve cell is subjected—all grades of chromatolysis, pigmentation, atrophy, and shrinkage of the nerve cells and processes, with larger or smaller pericellular spaces. Numerous capillaries, with thickened walls and perivascular spaces, are seen scattered throughout the various parts of the section. The actual size of this section is 8 mm. wide at the point opposite *d*, 8.5 mm. long, and 6 2-3 microns in thickness. At the points *a* and *b* are seen the Betz cells, shown under a magnification of 1,400 diameters in Plates XII, Fig. 35, and XII, Fig. 36. The former of these is a photomicrograph as stated, under a magnification of 1,400 diameters of the nerve cell, indicated by the letter *a* in Plate XI, Fig. 34. By referring to this latter plate, and then to Plate X, Fig. 29, we can locate almost the exact position of this cell in the cortex of Brain B. The cell-body is seen shrunken and deformed, lying in a pericellular space partially surrounding it. The nucleus is eccentric, being crowded over to the extreme edge of the cell-body on the right side. It is also shrunken and indistinct, but contains a well-marked and prominent nucleolus. The apical process above and to the right appears quite sharply deflected at a point but a short distance from the base, but this is not really the case, as the portion from *a* to the edge of the plate is one of the walls of a blood vessel, the opposite wall not being seen in this plate at all. At the point *a*, this apical dendritic process appears to come in direct contact with this portion of the wall of the blood vessel, and is there lost to view. The other dendritic processes—seven in all—are shrunken, and contain a small amount of diffuse chromatin. Within the cell-body the chromatin is considerable in amount, and quite generally diffused in fine granules throughout the cell-body, lacking any definite structural arrangement. There is a somewhat greater amount at the base to the left and below the nucleus, and also at the upper part of the cell-body. Above and to the left of the nucleus, where the cell-body appears most pale, and extending into the base of the dendritic processes here, is a considerable mass of yellowish pigment. Numerous neuroglia cells are seen in the vicinity of this Betz cell. Plate XII, Fig. 36, is also a photomicrograph, magnified 1,400 diameters, of another of these Betz cells from the point *b* of Plate XI, Fig. 34. Here there is but little diffuse chromatin, confined principally to the base of the dendritic process given off on the right. All the lower part of the cell-body, excepting this portion, contains palely yellow pigment. The nucleus is displaced almost to the extreme edge of the cell-body, and lies just below the two neuroglia cells seen at *a*. The nucleolus, on this account, can not be determined at this plane. On microscopic examination the nucleus is found to be shrunken and indistinct. Numerous neuroglia cells are seen upon and in the immediate vicinity of this cell. The basal dendritic process *b*, which is but faintly seen here, owing to its lying in a somewhat lower plane, has eight neuroglia cells in close apposition to it. The dendritic process on the right can be traced for some distance, is somewhat shrunken, and contains no chromatic substance. Another small dendritic process is given off from the base opposite the point *c*, and being in a lower plane, only its base is seen here. It is colorless, and extends but a short distance from the cell-body. The apical dendritic process contains no chromatin, and as seen, has several neuroglia cells surrounding it a short distance from its base. A large pericellular space almost entirely surrounds the cell, excepting at the base to the left. Plates XII, Fig. 37, and XII, Fig. 38, are photomicrographs of the same magnification (1,400 diameters), taken from an adjacent section of the same block as the two preceding plates, and are also 6 2-3 microns in thickness. This section was prepared in exactly the same way, with the exception that methylene blue was used as the stain instead of methylene violet. This will be at once apparent upon noticing the neuroglia cells, which are here pale and washed out, many to the point of complete decolorization, so as not to be seen at all. The Betz cell in the center of Plate XII, Fig. 37, is seen surrounded by a large pericellular space. All the processes, four in number, including the apical dendritic process, are pale, and terminate but a short distance from the cell-body. The nucleus is eccentrically situated near the wall at the left, is small, and contains a large nucleolus, but no nuclear network. Within the cell-body finely granular chromatic substance is found at the base below and to the right of the nucleus. There is no pigment present. This cell presents an advanced stage of partial chromatolysis. Above and to the right is an almost indeterminable mass, which, under the 1-12 inch oil-immersion objective, appears to be the remnant of a capillary, surrounded by a large perivascular space. Other cells are seen here in various stages of disintegration, surrounded by large pericellular spaces, some containing none or only the remnants of den-

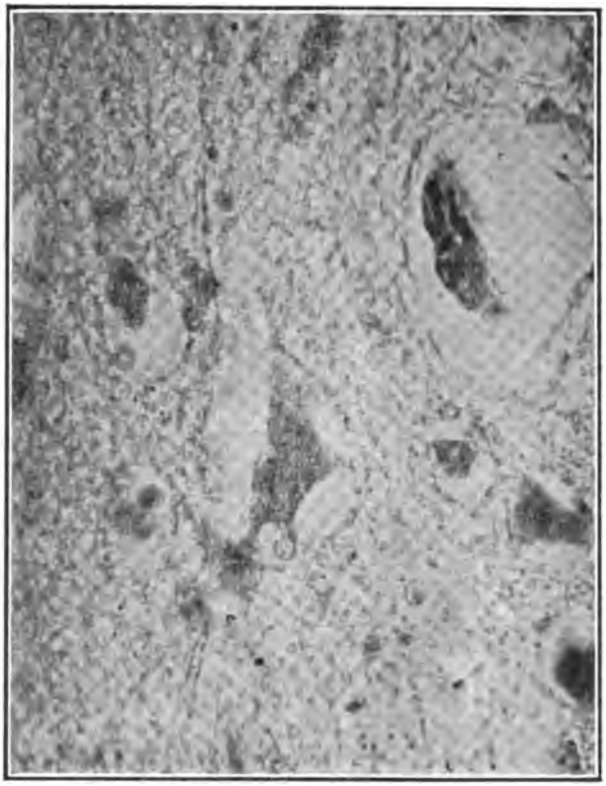

Plate XII, Fig. 37.

dritic processes, indistinct and shrunken nuclei, some distinct nucleoli and others none at all, and all in a more or less advanced state of chromatolysis, with but little of the diffused stained chromatic substance present. The neuroglia cells that are visible at all are pale and poorly stained, while many are almost or completely decolorized. Plate XII, Fig. 38, is a photomicrograph of a Betz cell on the same section and but a short distance from the cells of the preceding plate, and under the same magnification (1,400 diameters). Here there is almost complete chromatolysis, there being but little finely granular, diffuse chromatic substance scattered in the cell-body, slightly more at the base than above the nucleus. The latter is small, rounded, centrally situated, and

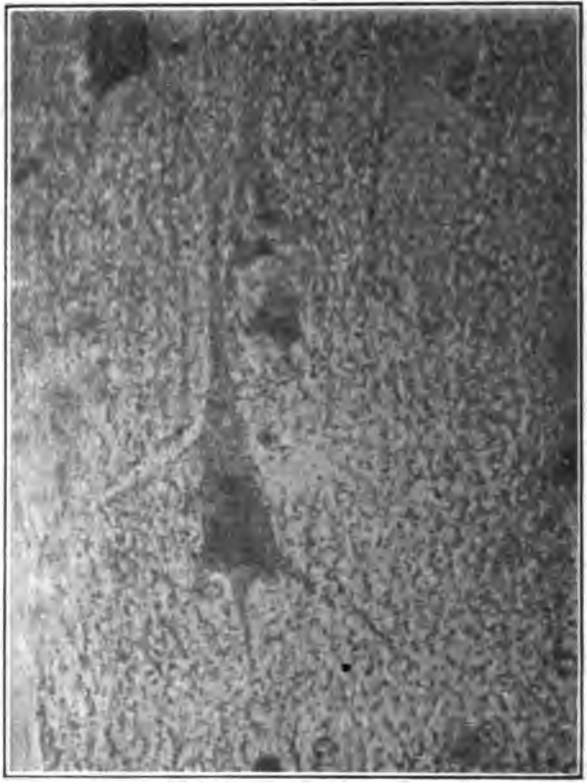

Plate XII, Fig. 38.

contains a large, rounded, distinct nucleolus. The three basal processes are narrow, pale, and terminate at no great distance from the cell-body. The apical process is pale and slender, extending for some little distance before coming to an end. Part of a pale, atrophied cell is seen to the right of the same. The ghosts of several neuroglia cells may be seen here, but most of them are completely decolorized.

Plate XIII, Fig. 39, as will be seen by referring to Plate X, Fig. 29, is from the lower portion of the upper third of the anterior central convolution, from the point marked O, and is magnified 14 diameters. This section, as already mentioned, is found to

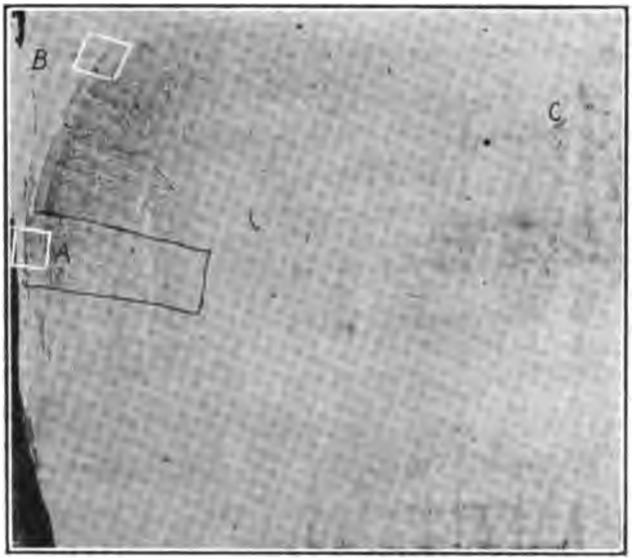

Plate XIII, Fig. 40.

vary somewhat in shape from that of the preceding sections shown in Plate XI, Fig. 32, and Plate XI, Fig. 34, being narrower and more pointed at the vertex. From the strip marked *a,* and enclosed in ink, the structure and arrangement of the nerve cells were carefully studied, and both nerve and neuroglia cell counts made and will be referred to again later. The irregularity and difference in thickness of the first layer in different parts is to be noted, being especially thick at *b,* where it almost presents the appearance of an artefact, but under a high power no derangement of the structure can be made out. At *a* the layer is seen to be very thin, owing to some of the surface of the cortex having remained adherent to and been stripped off with the thickened pia mater. Large perivascular spaces are to be especially noticed in the subcortical portion of the section. Here is to be noted the almost entire absence of the Betz or giant pyramidal cells in the lower part of the second layer, although some large pyramidal cells are to be found, they are not, however, so large nor are they arranged in such distinct groups as in the region higher up. Under higher magnification the remnants of some of these Betz cells are seen in various stages of necrosis and disintegration. The cortex lacks the distinct striated appearance seen in the sections of the normal cortex. This layer passes indistinctly into the third or spindle cell layer, which in turn is lost in the white medullary substance below. Numerous capillaries are seen, many with thickened walls and perivascular spaces throughout the section. This section was fixed in 95 per cent alcohol, sectioned 10 microns in thickness, stained with methylene violet, with other technique similar to that of all the previous sections. The actual size of the section is 9 mm. at its greatest width and 13 microns in length. The strip *a* varies from that of Plate XI, Fig. 33, only in the number and arrangement of the cells in the second layer. At this point the three layers are respectively .20 mm., 2.00 mm., and .90 mm. in thickness. The first layer contains some few scattered irregular nerve cells and numerous neuroglia cells. The second layer contains small pyramidal cells above, increasing in size until a little above the middle of the layer is a strip some .30 mm. in width, in which are to be seen large pyramidal cells arranged singly or in irregular groups. Some of these cells almost approach the giant pyramidal cells in size. Many of them show but a slight shrinkage and contain an almost normal amount of chromatic substance. The chromophilic bodies, however, are not as numerous or as large as in the normal brain. Other cells show a complete absence of chromophilic bodies, but contain the chromatic substance diffused throughout the cell in minute particles. Still others are pale, and contain but little chromatic substance. The nuclei are larger, less eccentric, and there is a less degree of atrophy of the cell-body and processes. Below this is a region of small pyramidal cells with only a few scattered larger pyramidal cells merging into the lower part of the layer, where large pyramidal cells are again seen scattered irregularly among the smaller cells. These large pyramidal cells are somewhat smaller than the giant pyramidal or Betz cells seen in the upper part of this convolution. These cells show more advanced pathological changes, in some cases marked atrophy and shrinkage with large pericellular spaces, atrophy of the dendritic processes, eccentricity and shrinkage of the nucleus, pigmentary deposits, complete absence of or but a small amount of diffuse chromatic substance. The third layer presents the same general appearance as in the preceding plate (XI, Fig. 33), with many cells much shrunken and atrophied and little or no chromatic substance within the same, and surrounded by pericellular spaces of varying size. Numerous large and small capillaries are seen scattered throughout this section, with thickened walls and surrounded by larger or smaller perivascular spaces. The entire depth of the cortex here is 3.10 mm., the second layer being slightly thicker than in Plate XI, Fig. 33. The nerve cells were found to average but 36.50 to the square millimeter of surface of the section here; less than in any of the other plates, whereas the neuroglia cells average 289 to the square millimeter of surface of the section, almost as great a num-

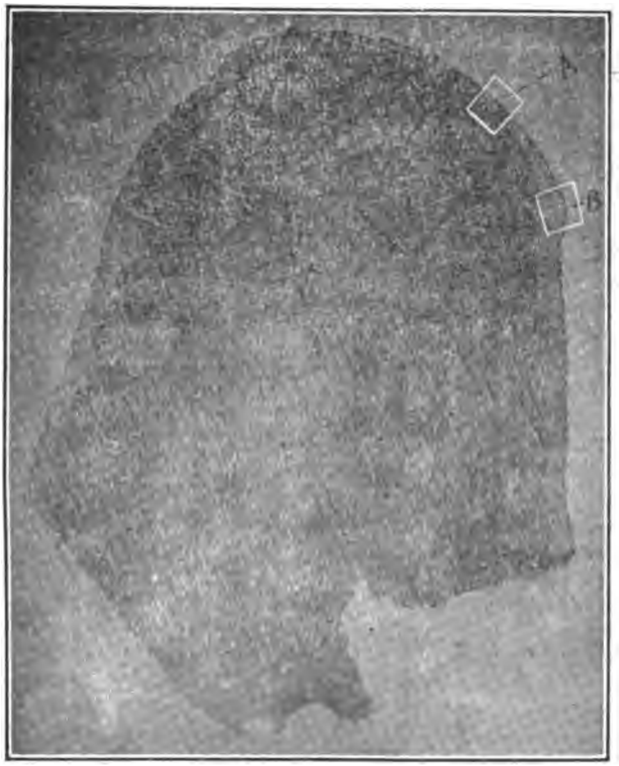

Plate XIII, Fig. 39.

ber as in the previous sections of the same thickness, ten microns, and stained with methylene violet. The most conspicuous difference between this and Plate XI. Fig. 33, higher up in the convolution, is the arrangement of the large pyramidal cells, the absence of the typical Betz cells, and on the whole less advanced pathological changes. Plate XIII, Fig. 40, is a photomicrograph magnified fourteen diameters of a section from the block B of Plate X, Fig. 29, and is thus seen to be situated a little above the middle of the posterior central convolution. This section is 6 2-3 microns in thickness, 8 mm. in length, and 9 mm. in width at the widest point, was fixed in 95% alcohol, stained with methylene blue, and other technique similar to that of all previously described sections. This section at the vertex, especially to the left, shows the striated appearance of cell arrangement very well, but upon the anterior and posterior aspects of the gyrus it is but indistinctly shown. The first layer is fairly uniform in thickness with broken spaces here and there, especially about the entrance of capillaries. This layer is somewhat thicker in certain places than in others. At *a*, for instance, it is thicker than at the vertex. This section has a broad, flattened vertex with considerable cortical area on both the anterior and posterior surfaces. The cortex opposite the angles *c* and *b* is somewhat thicker than elsewhere. This mechanical arrangement admits of a large, broad mass of fibres from various parts of this and the adjacent regions of the brain, leaving from and entering into relations with the cells of this portion of the cortex. The section is broken (an artefact) at the left and numerous capillaries are found scattered throughout all the cortical layers and also the white medullary center. The segment represented in Plate XIII, Fig. 41, was taken from a point corresponding to *a*, but from an adjacent section of the same block, and is magnified 100 diameters. It is from relatively the same position as the segment shown in Plate VII, Fig. 19, the strip *a* of Plate VI, Fig. 18, of the normal brain, but, as will be seen by referring to the latter, the shapes of the two sections are quite different. The distribution and arrangement of the cells in this plate is somewhat different from that in Plate VII, Fig. 19, also. In this latter, as already described, some large pyramidal cells are irregularly distributed in parts of the upper half of the second layer, followed by a narrow region made up almost exclusively of small pyramidal cells to be followed by larger pyramidal cells in the lower portion of this layer. In this plate, however, there are but few of the larger pyramidal cells in the upper portion of the second layer, there being practically only small pyramidal cells in this upper portion with the larger pyramids mostly in the lower portion of this second layer. The cell seen in the lower part near the center (*a*, Plate XIII, Fig. 41), approaches in size to the Betz cell type. The first or superficial layer here contains nothing but capillaries and neuroglia cells and is .25 mm. in thickness. The second or pyramidal cell layer is 1.40 mm. in thickness and shows not only numerous capillaries with irregular and thickened walls, but also the cells in various pathological conditions. Chromatolysis is complete in many of these cells as in the large pyramidal cell marked *a* for instance, where only traces of minute finely powdered chromatin can be seen in some parts of the cell-body. Here also the nucleus is indistinct, with a well marked nucleolus, and the basal processes are much atrophied, terminating but a short distance from the cell-body. A small pericellular space is seen at the base of this cell-body. Many cells show eccentricity of the cell nucleus, shrinkage of the cell-body, various grades of chromatolysis, pigmentation, atrophy, and distortion of the dendritic processes and are surrounded by pericellular spaces. The capillaries are numerous, have thickened and irregular walls, and are surrounded by perivascular spaces of varying size and extent. Numerous neuroglia cells are also interspersed about the cells and capillaries in this layer. The third or spindle cell layer is only partially seen in this plate, and is about .90 mm. in thickness (only upper portion shown in Plate XIII, Fig. 41), and is similar to that in preceding plates of the motor region, and contains for the most part shrunken and irregular spindle and polygonal shaped cells, with numerous neuroglia cells and capillaries interspersed among them. Most of these cells show the various pathological processes mentioned above for the pyramidal cells. The cortex here thus measures 2.55 mm. in thickness, almost the same as for the two upper layers in Plate VII, Fig. 19, but here the third layer is .90 mm. in thickness as compared to .60 mm. in the above mentioned plate of the normal brain. Nerve and neuroglia cell counts were made here with the result that an average of 59.91 nerve cells and 134.37 neuroglia cells were found to the square millimeter of surface of the section as compared to an average of 185.50 nerve cells and 109.20 neuroglia cells for the same area in the strip of cortex represented in Plate VII, Fig. 19, of the corresponding region of the normal brain. Both sections were 6 2-3 microns in thickness and both stained with methylene blue. The difference in nerve cells is very marked, whereas the difference in number of neuroglia cells is not so great, and as the technique was identical throughout in the preparation of the two sections, the marked pathological process shown in this latter, it seems to the writer must be attributed as the cause of this difference to a large extent at least.

*Parietal Region.*—Plate XIV, Fig. 42, is a photomicrograph magnified fourteen diameters, of the parietal region from the point H. in Plate X, Fig. 29, this being a part of the supra-angular gyrus and from the same relative part of the gyrus as the corresponding plate (VII, Fig. 20) of the normal brain. The section was fixed in 95% alcohol, stained with methylene violet, and treated otherwise as the preceding plates. This section measures 6 mm. in width, 16 mm. in length, and is 6 2-3 microns in thickness. Its shape is long and narrow and quite different from that of Plate VII, Fig. 20. At the vertex of the convolution the first layer has geen cut away with the paraffin in preparing the block for sectioning serially with a Minot microtome, and, of course, this condition is an artefact. The first layer is seen to be fairly uniform in thickness with this exception. In the second layer it is difficult to determine the well marked striated arrangement of the cells, as seen in Plate VII, Fig. 20. Numerous perivascular and pericellular spaces are seen with or without the capil-

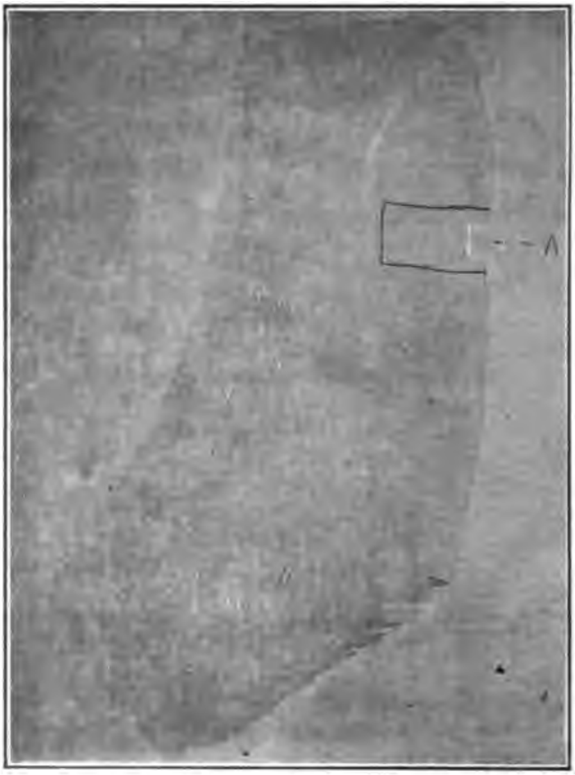

Plate XIV, Fig. 42.

laries or cells, as the case may be. The third layer is indistinct and fades away into the white medullary center which also contains many pericellular and perivascular spaces. No doubt the fixation in 95% alcohol has produced some shrinkage, but by no means all, as the plates of the occipital convolution just posterior to this show a much less degree of shrinkage than is here present and not only every block taken from this brain was fixed in exactly the same way, but also some of the blocks from Brain A. At *a* and surrounded by ink lines is the segment of the section shown under a magnification of 100 diameters in Plate XIV, Fig. 43, and as is here seen is taken from the lateral aspect near the vertex, instead of from the vertex as in the corresponding plate of the normal cortex (Plate VII, Fig. 21). The first layer averages .25 mm. in thickness and contains numerous capillaries with thickened walls, and tortuous course with larger and smaller perivascular spaces. Numerous neuroglia cells are scattered throughout this layer. The second layer is here 1.50 mm. in thickness. Here, too, are seen numerous capillaries with thickened walls, irregular course, and large perivascular spaces. In the upper half of the layer the small pyramidal cells gradually are intermingled with larger pyramidal cells as the lower portions are reached, until just above *a* the largest pyramids of the section are seen singly or in groups in a comparatively narrow zone. Below and opposite *a* is a narrow zone, some .25 mm. in width in which there are practically no large pyramidal cells. Below this again is a very narrow zone at the lower part of this layer in which some few large pyramidal cells are seen scattered among the smaller and irregular cells. These cells present all stages of chromatolysis, pigmentation, shrinkage of the cell-body and processes and irregularity in direction of the latter, eccentricity and shrinkage of the nucleus and larger and smaller pericellular spaces. Numerous neuroglia cells are scattered throughout the layer. The third, or spindle cell layer, measures scarce .50 mm. in thickness and contains for the most part shrunken and irregular spindle cells in larger or smaller pericellular spaces, in many instances in various stages of chromatolysis. Numerous neuroglia cells in many cases appearing in direct contact with the cell-body are seen in this layer, as well as in the above layer. The vascular changes are also similar. The entire depth of the cortex here is thus 2.25 mm., somewhat less than in Plate VII. Fig. 21, but about the same as on the lateral aspect of this latter plate. Nerve and neuroglia cell counts were made here in the same manner as in previous sections, and it was found that there was an average of 68.63 nerve cells and 192.70 neuroglia cells to each square millimeter of surface of the section as compared to 104 nerve cells and 180.90 neuroglia cells in the corresponding region of Brain A, both sections being of the same thickness and stained in the same manner with methylene violet.

*Temporal Region.*—Turning to the region below this we come to the temporal region represented here by a section from the first temporal convolution near its anterior extremity, from the

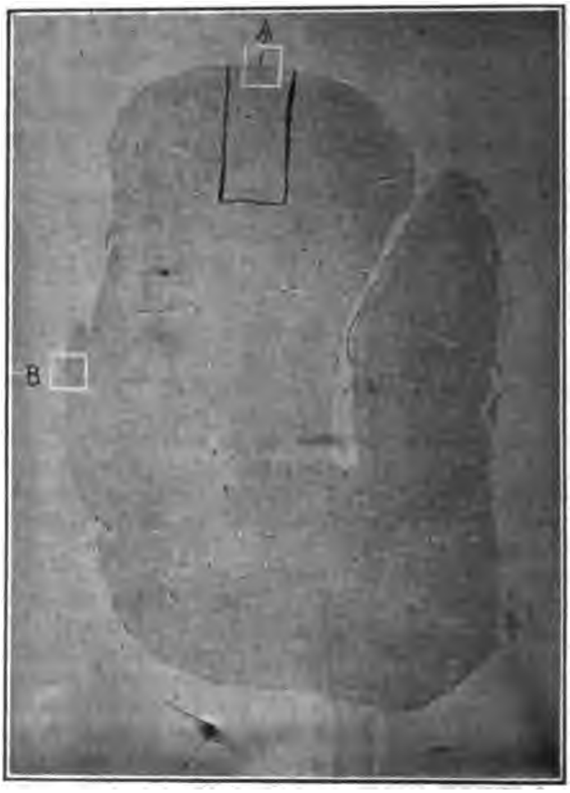

Plate XIV, Fig. 44.

point *I* in Plate X. Fig. 29, and shown under a magnification of fourteen diameters in Plate XIV. Fig. 44. This convolution is also much narrower than the corresponding convolution of *Brain A*, seen in Plate VIII, Fig. 22, thus having a somewhat dissimilar mechanical arrangement. To the right is seen the first temporal fissure and a portion of the second temporal convolution. The convolution measures 5.5 mm. in width and 8 mm. in depth from the vertex to the point on the level with the bottom of the first temporal fissure and is 6 2-3 microns in thickness. The convolution at this point is seen on the right to curve around and fuse with the second temporal convolution, being free above at *b*, which is the lower boundary of the fissure of Sylvius at the beginning of the posterior branch. At *a* and enclosed in ink lines is the segment of the section shown in the following plate under a magnification of 100 diameters. The first or superficial layer is quite uniform in thickness, broken in several places, especially above *b* on the superior surface. The second layer shows the striated arrangement of the cells only very indistinctly at some few points and contains many pericellular and perivascular spaces, some with and others without contents. The third layer is indistinct and fades into the white medullary center which is narrow and passes inferiorly over to connect on the right with that of the second temporal convolution.

In Plate XV, Fig. 45, strip *a* of Plate XIV, Fig. 44, under a magnification of 100 diameters, the pathological conditions are seen to be well marked. The section is 6 2-3 microns in thickness and stained with methylene violet. Here the first layer averages about .20 mm. in thickness and to the right a fragment of a blood vessel is seen at the surface, but penetrating the cortex in a different plane. In the center and reaching into the second layer is a large capillary with thickened walls, with larger caliber above and smaller below, being somewhat funnel shaped and surrounded by a large perivascular space. Some smaller fragments of capillaries and neuroglia cells are scattered about in the layer. The second or pyramidal cell layer is 1.50 mm. in thickness and presents very much the same general plan of arrangement as the corresponding region in the normal brain, as seen in Plate VIII, Fig. 23, and also in the region of the first frontal convolution, as seen in Plate II, Fig. 3, and XI, Fig. 31. Small pyramidal cells almost exclusively are seen in the upper part of the layer below this gradually increasing in size to the middle of the layer. Then opposite *a* is a narrow strip some .20 mm. in thickness in which there are small pyramidal cells almost exclusively. Below this again and in the lower part of this layer are larger pyramidal cells, intermingled with smaller pyramidal and irregular shaped cells. Here, too, the majority of the cells are seen shrunken and surrounded by larger and smaller pericellular spaces. Various stages of chromatolysis, shrinkage and eccentricity of the nucleus and atrophy of the cell processes are to be observed. The blood vessels show the same pathological conditions as in the upper layer and numerous neuroglia cells are interspersed thickly everywhere.

The third layer is .80 mm. in thickness and presents the same